Acta
OTO-LARYNGOLOGICA

S U P P L E M E N T U M 175

OBJECTIVE AUDITORY TESTS ON NON-COOPERATIVE CHILDREN

A Follow-up Examination of 50 Newborn Infants and Pre-School Children with Suspected Hearing Loss

BY

ERIK WEDENBERG

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO LARYNGOLOGICA

SUPPLEMENTUM 175

FROM THE DEPARTMENT OF OTOLARYNGOLOGY (HEAD PROFESSOR CARL AXEL HAMBERGER M D) AND THE DEPARTMENT OF AUDIOLOGY (HEAD ASSOCIATE PROFESSOR LENNART HOLMGREN M D) KAROLINSKA HOSPITAL STOCKHOLM FROM THE DEPARTMENT OF TELEGRAPHY AND TELEPHONY (HEAD PROFESSOR TORBERN LAURENT) THE SPEECH TRANSMISSION LABORATORY (HEAD ASSOCIATE PROFESSOR GUNNAR FANT) ROYAL INSTITUTE OF TECHNOLOGY STOCKHOLM

OBJECTIVE AUDITORY TESTS ON NON-COOPERATIVE CHILDREN

BY

ERIK WEDENBERG

A FOLLOW UP EXAMINATION OF 50 NEWBORN INFANTS AND PRE SCHOOL CHILDREN WITH SUSPECTED HEARING LOSS

STOCKHOLM 1963

Contents

Introduction

In October, 1955, the author performed auditory tests on a presumably normal series of 20 newborn aged from one to 10 days. The results of this study have been published in an earlier article*, to which the reader is referred for a list of references. The present article is concerned with a follow-up study of this group and a corresponding study of 30 children with suspected impairment of hearing, performed during the period 1955—58. In the case of both groups a preliminary assessment of the hearing was made by means of the auro-palpebral reflex (APR) and the waking tests (see below), the control study relied on play audiometry, or, where this was not possible, informal tests.

Technique

APR and waking tests — These tests require a free-field presentation of tones, with extremely rapid build up. For this purpose a special audiometer has been designed.** The instrument (Fig. 1) covers the frequency range 500—4000 cps in half-octave steps, and the sound intensity is adjustable in 5 dB steps. A maximum of 120 dB relative to 0.0002 dynes per sq. cm is obtainable 50 cm from the loudspeaker. The build-up time is about 2 msec.

To enable all the values in the audiograms to be compared the calibration of this free field audiometer was corrected in such a way that the APR and waking level are also referred to the normal hearing threshold.

The preliminary hearing test included (i) threshold determination of the APR, that is, the rapid and distinct closing of the eyelids which is brought about by contraction of the orbicularis oculi muscle in response to intense acoustic stimulation under certain conditions, and (ii) determination of the intensity at which it is possible to awaken the child.

The tests were performed in a nursery room at the maternity or pediatric ward during periods when the surrounding noise level was judged to be reasonably low. No further precautions were taken to insure a silent testing environment. (Fig. 1.)

* Auditory tests on new born infants. Erik Wedenberg. Acta Oto-Laryngologica, vol. 46, fasc. 5, Sept.-Okt. 1956.

** By Mr. Bertil Johansson at the Department of Telegraphy and Telephony, The Royal Institute of Technology, Stockholm.

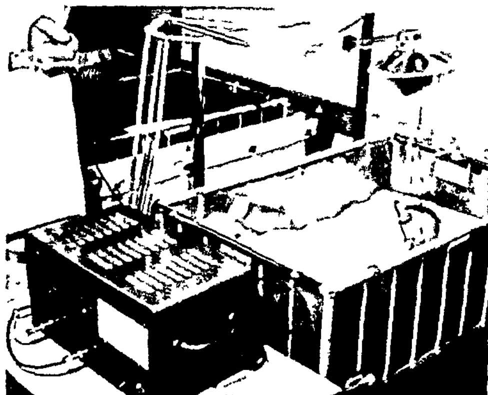


Fig. 1. Arrangement for the audiometric test on new-born infants.

During the tests the infant lay on its side in its cot, as a rule sleeping. Since in this position one ear was covered by the pillow only one ear was tested at a time. With the applied frequencies of 500—4000 cps it was possible in all the normal subjects to elicit the APR at a level of 100—115 dB above the threshold for normal hearing. The following difficulties were encountered in determining the reflex threshold when sleeping and sometimes when awake some of the children did not respond to the loudest tone (115 dB). (i) All those who did not respond with the APR *in the sleeping state* were so deeply asleep that it was not possible to elicit the lid reflex by tactile stimulus. They were thus in anaesthesiologic terms in the lower plane of the second stage or in the first plane of the third stage of anaesthesia (Fig. 2). If they were roused to the point at which the lid reflex could be provoked by tactile stimulus then it was also possible to elicit this acoustically. (ii) Those children who did not respond with the APR *in the waking state* were all preoccupied — for instance with [illegible] some other display of discomfort. The absence of a reaction in [illegible] was probably due to reflex inhibition.

The APR threshold curve for the group of normal subjects bears a close resemblance to the curve for the stapedius reflex recorded in tests on adults with normal hearing (Jepsen 1955) (Figs. 3 and 4). From the practical [illegible] the two reflexes [illegible] (Fig. 3). For instance [illegible]

[illegible]

Signs of anæsthesia
(ad m T Gordh)

Intravenous anæsthesia

		Stages	Lid reflex	Skin reflex	Respiration Diaphr Int cost	Pupils	Approximate levels for abolition of reflexes
I		Conscious Increasing drowsiness					
II		Nonconscious Eyeball movements Lidreflex retained					Lidreflex
III	1	Lidreflex abolished Skinreflex retained					Skinreflex
	2	Skinreflex abolished Respiratory depression sets in					Corneal reflex
	3	Shallow thoraco abdominal respiration Contracted pupils					Glottic reflex Pup light reflex
	4	Abdominal respiration Cyanosis Dilating pupils Insensitive to light					
IV		Respiratory paralysis Exitus					

Fig 2

afferent (acoustic nerve) and efferent part (facial nerve) of the reflex arcs are common to both, though the centre of the stapedius reflex is at a lower level of the brain-stem (pons) than the APR (reticular formation) The APR threshold curve for the presumably normal newborn subjects (Fig 3) should thus indicate normal hearing In the individual case, however, the fact that the APR has been elicited at a certain frequency and "normal" intensity is no proof that the hearing for the frequency in question is normal In cases of hearing impair-

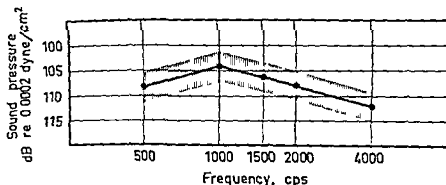


Fig 3 Mean threshold curve for the auro-palpebral reflex of new born infants

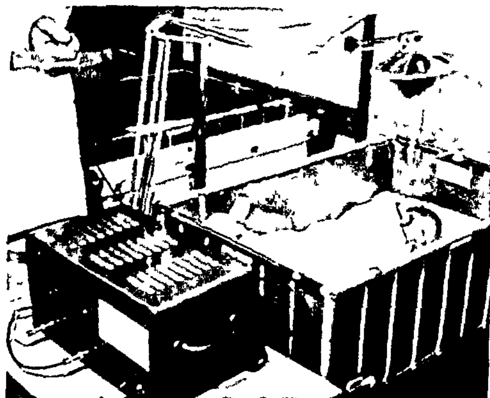


Fig. 1. Arrangements for the auditory tests on new-born infants.

During the tests the infant lay on its side in its cot, as a rule sleeping. Since in this position one ear was covered by the pillow only one ear was tested at a time. With the applied frequencies of 500—4000 cps it was possible in all the normal subjects to elicit the APR at a level of 100—115 dB above the threshold for normal hearing. The following difficulties were encountered in determining the reflex threshold: when sleeping and sometimes when awake some of the children did not respond to the loudest tone (115 dB). (i) All those who did not respond with the APR *in the sleeping state* were so deeply asleep that it was not possible to elicit the lid reflex by tactile stimulus. They were thus, in anaesthesiologic terms, in the lower plane of the second stage, or in the first plane of the third stage, of anaesthesia (Fig. 2). If they were roused to the point at which the lid reflex could be provoked by tactile stimulus then it was also possible to elicit this acoustically. (ii) Those children who did not respond with the APR *in the waking state* were all preoccupied — for instance with [illegible] or some other display of discontent. The absence of a reaction in [illegible] was probably due to reflex inhibition.

The APR [illegible] curve for the group of normal [illegible] bears a close resemblance [illegible] the stapedius reflex recorded in tests on adults with normal hearing [illegible] 1955) (Figs. 3 and 4). From the purely anatomical standpoint the two reflexes [illegible] similarities (Fig. 5). For instance, th[illegible]

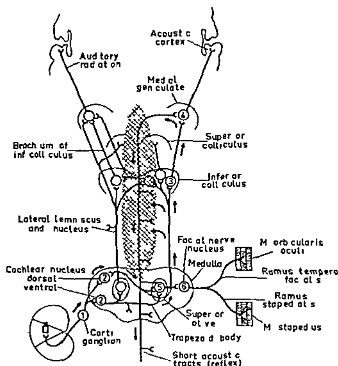


Fig 5 The afferent acoustic pathways (based mainly on cat) The order of each neuron is indicated by a number (From Hallowell Davis 1951) In this figure are inscribed the supposed ... and the ... and m ... umber 6 ... l reflex

1 2 $<^{3}_{4}>$ 5 6 ramus tempero-facialis

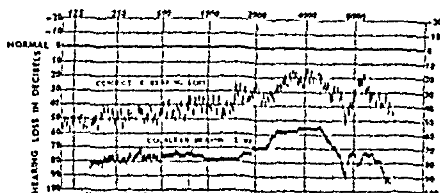
the hearing is impaired, without performing standard threshold determinations (Barr) The child s response to different sounds is observed and evaluated in relation to that of the normal child to the same sound, at the same developmental age The informal tests fall into three groups with respect to the nature of the sound used

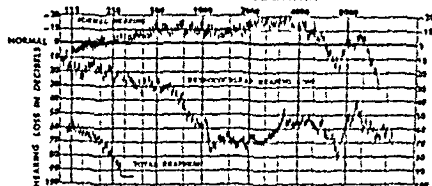
(i) Percussion noises (the sound of cow bells, drums and triangles),
(ii) Meaningful noises (the rustle of paper, sounds from toy animals),
(iii) Speech (tests with the spoken or whispered voice)

	Normal hearing	Cochlear hearing loss	Conductive hearing loss Retrocochlear hearing loss	Total deafness Severely hard of hearing
APR	+	+	—	—
Waking ≦70-75 dB*	+	—	—	—
Waking >70-75 dB	+	+	+	—

Fig 6 Prospective schedule of different kinds of hearing loss

* Sound pressure level





Figs. 7 and 8. Examples of Békésy audiograms showing different [illegible]

The informal tests can provide a fairly good impression of the hearing of the subject. They were used in 6 cases in which the low intelligence of the child ruled out the use of play audiograms (cases 1, 12, 20, 22, 26, 27).

Case material

The 50 subjects on whom hearing tests were performed consisted of 20 children with supposedly normal hearing and 30 with suspected impairment of hearing.

(1) *The normal group.* — The 20 children composing the normal group who were examined during their first 1—10 days of life, have now grown up and are more than 5 years old so that a check of the APP and waking tests performed at birth could now be made by conventional audiometry.

(2) *Group with suspected hearing impairment* — The same hearing tests were performed on the 30 children suspected of having impaired hearing The children had been admitted to the Ear, Nose and Throat Department or the Department of Paediatrics at the Karolinska Hospital, and at the time of the examination they ranged in age from a few days to $4\frac{5}{12}$ years For none of the children could the play audiograms be made at the original tests Twenty-five of the children were too young to cooperate in such tests (less than 2 years), and the other 5 who were more than 2 years were unable to cooperate owing to low intelligence, spasticity or immaturity

Follow up play audiograms were subseqvently recorded for most of these subjects In 20 cases altogether control audiograms were recorded in 6 informal tests were made, and 4 patients died The age distribution was extremely wide

Age at first examination (months)	< 1	1—3	3—12	12—24	> 24	Total
Number of subjects	9	3	4	9	5	30

As noted above, the group consisted of 30 subjects with suspected hearing damage It is a very heterogeneous group with respect to medical diagnosis (Table 1) One of the questions of interest was whether any hearing impairment was solitary or accompanied by other discorders No less than 9 of the children were of low intelligence — most of them older children, 5 of them being more than 12 months at the examination Since, from the standpoint of aetiology and diagnosis the cases differ widely from one another, each case will be dealt with separately (pp 14—26)

Results of follow-up tests

The normal group — This group on whom the hearing tests were performed in October 1955, consisted of 20 children Difficulty was encountered in performing the control tests on some of the children owing to removal to other parts of the country In 10 cases play audiometry was performed, for all but one of whom the hearing was normal (Fig 9, p 12 audiogram S P)

In the exceptional case a decrease of 20 dB was noted for one ear in the range 125—500 cps This reduction may, however, be ascribed to otitis, which had affected this ear 14 days prior to the examination, and traces of which were still noted on the occasion of the test

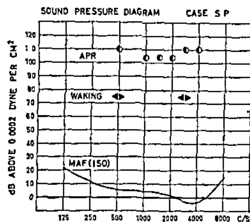


Fig 9 A Auditory tests in a normal hearing child with APR and Waking Thresholds recorded in dB above 0 0002 dynes per sq cm

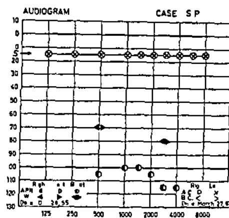


Fig 9 B Same values as in fig 9 A in audiometric representation i e with reference to normal threshold of hearing Subject s air conduction from threshold follow up examination added For details see text

The sound pressure levels necessary for provoking the APR and the waking effect are shown in Fig 9 A case S P Otologists, however, are in general more familiar with the representations of the results in audiograms In fig 9 B the levels for the same patient, S P, therefore are shown in an audiogram, together with a play-audiogram of a later date It should be observed that both the APR and the waking effect are plotted relative equivalent normal threshold of hearing

In fig 9 A the MAF (minimum audible field) according to the ISO recommendation R 226 is indicated for comparison This threshold curve has also been used when transferring the data for APR and waking thresholds into the audiogram The ISO curve is obtained for adults under free field conditions with the observer facing the loudspeaker The children, on the other hand, have been tested in bed lying on their side with one ear covered by the pillow This implies somewhat different conditions in comparison with the ISO curve, concerning difference of incidence which may give a deviation of about 5 dB, and further a difference in diffraction due to the skull and the acoustical conditions of the ear canal and the middle ear Concerning the special position of the child in bed it can be considered that the absorption of the bed for the frequency range used, 500—4000 c/s, is rather high

It may also be mentioned that the difference between APR and waking thresholds presented directly as sound pressure levels, and presented relative MAF values, are confined to a range of about 5 dB Therefore it could be discussed whether to abstain from any corrections or not

Concerning the following figures it may be added that in cases 11, 17, 28 and 30 the pure tone audiograms could only be presented as binaural thresholds in a free field measurement

The group with suspected hearing impairment (Table 1 and 2) — The findings in this group varied widely and the 30 cases will therefore be reported individually (pp 14—26)

Table 1 Group with suspected hearing impairment

Case no	General diagnosis	Age
1 G P ♀	Asphyxia neonatorum c athlectas pulm immaturitas + cataract + *vitium org cord. cong.*, + *haemorrhagia intracran*, + surditas?	
2 P S ♂	Dysontogenesis auris sin et mandibulae sin + surditas?	
3 O.M ♀	Mikrocephalia + surditas?	
4 W O ♂	Surditas hereditaria?	
5 R ♀	" "	< 1 month
6 H A ♀	" "	
7 L. ♂	" "	
8 J T ♂	Morbus haemolyticus neonatorum + icterus nucleosus + surditas?	
9 B ♀	*Morbus haemolyticus neonatorum + surditas?*	
10 J A ♂	Surditas?	
11 O ♂	Morbus haemolyticus neonatorum (A immunisation) + *hydrocephalus incertae causae, + surditas?*	1—3 months
12 E. ♂	Immaturitas, + encephalopatia + surditas?	
13 P A ♀	Status post morbus haemolyticus neonatorum cum icterus nucleosus + bronchopneumonia + surditas?	
14 S ♂	Encephalopatia + status post osteomyelit + amaurosis congenita bilat, + surditas?	
15 W E ♀	Aplasia nuclei facialis — abducens — hypoglossus + agenesia dig II + V sin et dig II dx + syndactylia dig II—III dx + bronchopneumonia + surditas?	3—12 months
16 E.L ♂	Haemorrhagia subarachnoidalis + paresis nervi fac dx + cystopyelit + rachitis + anæmia sec + surditas?	
17 W E ♀	Paralysis cerebralis + surditas?	
18 A B ♀	Surditas?	
19 M L ♀	Surditas hereditaria?	
20 J C ♀	*Oligophrenia + surditas?*	
21 R.E ♀	" "	1—2 years
22 S A ♂	Encephalopatia progressiva + surditas?	
23 S L. ♀	Surditas?	
24 T C ♂	Surditas?	
25 H P ♂	Debilitas psychica + surditas?	
26 H I ♂	Oligophrenia + amaurosis + surditas?	
27 K T ♂	Oligophrenia + surditas?	
28 A M ♂	Oligophrenia + epilepsia + surditas?	> 2 years
29 S R ♂	Surditas	
30 J K ♀	Debilitas psychica + surditas?	

CASE 1 G P ♀ b 16 4 56

Heredity * — Not remarkable

Pregnancy and delivery normal

Peri-natal status — Limp, respiration did not begin for 4 min, incubator 17 4 56 Moro embrace and grasp reflexes lively

General diagnosis — Asphyxia neonatorum with atelectasis, immaturity, cataract, congenital heart defect, intracranial haemorrhage, hearing impairment?

Original hearing test — At 3 weeks hearing normal

Follow up test — At $4\frac{9}{12}$ years, informal tests hearing normal

Comments — No play audiogram owing to oligophrenia

CASE 2 P S ♂ b 11 4 56

Peri natal status — Deformity of left side of face with left external ear and auditory canal absent Mother did not think the child reacted to sound

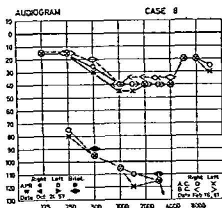
General diagnosis — Defective development of left ear and mandible, deafness?

Original hearing tests — At 3 weeks, right ear hearing normal, left ear conductive loss = or > 40 dB

Follow-up test — At $4\frac{10}{12}$ years right ear, hearing normal, left ear, conductive loss, 60 dB

Comments — During the test on the deformed left ear the normal right ear was masked with an ear plug of sponge rubber and a rubber cover Left ear no APR Waking test with 110 dB positive at 500 and 3000 cps According to the chart (Fig 8) the absence of APR and a positive waking test at intensities greater than 70—75 dB indicate either a conductive or a retrocochlear loss, in this case of a deformed ear the former is by far the more likely In both forms of impairment the loudness increases parallel with the loudness perception of a person wth normal hearing, so that waking at 110 dB would indicate a hearing loss of 110 less 70, or 40 dB The follow-up audiogram recorded on 6 2 61, however, showed a conductive loss of 60 dB The discrepancy may be ascribed to imperfect masking of the normal ear at the original test

* In the following cases heredity pregnancy delivery and peri natal status were normal or not remarkable unless otherwise stated



Dash dotted curve Stapedius reflex bilat. Feb 16 61

CASE 8 J T ♂ b 9 10 57

Heredity — Third child of Rh immunized mother

Peri natal status — Jaundice on third day, opisthotonus posture Bilirubin 22 3 mg per 100 ml No Moro embrace or grasp reflexes The reflexes gradually recovered

General diagnosis — Neonatal haemolytic disease, nuclear jaundice, hearing impairment?

Original hearing test — At 14 days bilateral retrocochlear loss

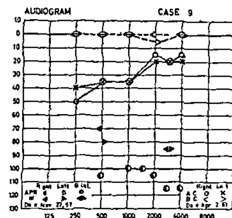
Follow-up test — At $2\frac{1}{2}$ and $3\frac{4}{12}$ years bilateral retrocochlear loss

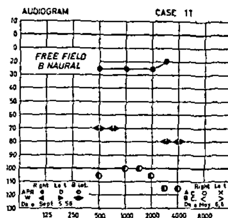
Comments — According to the chart in Fig 8 the absence of APR but waking at higher intensities than 70—75 dB at 500 and 3000 cps indicates either conductive or retrocochlear loss The former is ruled out by the ear examination which disclosed normal conditions, the diagnosis is therefore retrocochlear hearing loss, and this is compatible with the localisation of the hearing defect in neonatal haemolytic disease, namely the dorsal and ventral nucleus in the medulla oblongata Since, in retrocochlear impairment, the loudness increases parallel with the loudness perception of the person with normal hearing, waking at 500 cps and 95 dB indicates a hearing loss of 95 less 70, or 25 dB, and waking at 3000 cps and 105 dB a loss of 105 less 70, or 35 dB At the examination at $2\frac{1}{2}$ years the child could perceive the *s*-sound well at several metres, this implies fairly good hearing above 4000 cps, beyond which frequency the main formants of the *s*-sound lie *In this case an audiogram could be recorded at the first test performed when the child was 14 days old*

CASE 9 B ♀ b 10 11 57

Heredity — Second child of Rh immunized mother

Peri natal status — Umbilical blood showed Meulengracht 1 24 Max. bilirubin 22 9 mg per 100 ml Three exchange transfusions On the fourth day, listless fed by tube Suspected cerebral damage





General diagnosis — Neonatal haemolytic disease, hearing impairment?

Original hearing test — At 16 days hearing normal

Follow-up test — At $3\frac{5}{12}$ years conductive loss in the lower frequencies, hearing normal in higher frequencies

Comments — The conductive loss was due to a common cold on the occasion of the test, both ear-drums were much retracted, when in good health the hearing was probably normal and the original finding of normal hearing would then have been correct

CASE 10 J A ♂ b 17 9 56

General diagnosis — Hearing impairment?

Original hearing test — At 6 weeks hearing normal

Follow-up test — At $4\frac{5}{12}$ years, play audiogram hearing normal

} Cf, fig 9, p 12

Comments — The mother had another, older child that was born with severe visual and hearing impairment due to German measles in the second month of pregnancy She now suspected total deafness in the last child and had attempted suicide The child and mother were admitted to the Department of Otolaryngology for hearing tests

CASE 11 O ♂ b 6 8 56

Peri-natal status — At 2 weeks, bilirubin 25 mg per 100 ml Mother's blood group O Rh-, her serum contained anti-A of immune type but not Rh anti-bodies, father's blood group A Rh+ Exchange transfusion at 4 weeks Child became brighter

General diagnosis — Neonatal haemolytic disease (A-immunisation), hydrocephalus of uncertain origin, hearing impairment?

Original hearing test — At 6 weeks hearing normal

Follow-up test — At $4\frac{9}{12}$ years, play audiogram hearing normal

Comments — Owing to the mental backwardness it was difficult to record a play audiogram In the screening procedure therefore, 25 dB was accepted as the threshold

CASE 12 E ♂ b 29 9 57

Pregnancy — The mother had taken 316 analgesic tablets since the third month

Delivery — Five weeks premature

Peri-natal status — Bilirubin 22 7 mg per 100 ml the first week, spasms, transfusion

General diagnosis — Immaturity, encephalopathy, hearing impairment?

Original hearing tests — At 8 weeks, hearing normal (Cf , fig 9, p 12)

Follow-up test — At 4 years, informal test. hearing normal

Comments — The patient was feeble-minded, some understanding of the spoken word The author asked the mother to whisper something behind the child's back On one occasion when the child was unhappy she whispered very softly "Do you want to bath?" He understood, brightened up and looked very gay, his despondent mood returned but when his mother whispered again his face lit up This was repeated several times with the same result When the author tried whispering the same thing behind his back, the child registered no change of expression Hence, the patient could distinguish between his mother's and the author's whispered voice When the discriminatory power is so good the hearing is almost certainly normal

CASE 13 P A ♀ b 28 7 55

Heredity — The second child of a Rh immunized mother

Peri-natal status — At 3 days Meulengracht 1 200, blood transfusion No Moro embrace reflex Opisthotonus posture At 4 days a further blood trans fusion Signs of brain damage

General diagnosis — Typical of state after neonatal haemolytic disease, nuclear jaundice, bronchopneumonia, hearing impairment?

Original hearing test — At 4 months severe bilateral hearing impairment with residual hearing (APR negative, wakening at 100—115 dB, 500—2000 c/s)

Follow-up test — Child died at 7 months

CASE 14 S ♂ b 2 1 58

Peri-natal status — Born with branchiogenic cyst

General diagnosis — Encephalopathy, status post osteomyelitis, congenital bilateral amaurosis, hearing impairment?

Original hearing tests — At 3 months hearing normal (Cf, fig 9, p 12)

Follow-up test — Child died at 2 years

CASE 15 W E ♀ b 6 12 57

Peri natal status — Bilateral paresis of facial, abducens and hypoglossal nerves and deformation of fingers of both hands The child lay with the eyes open, *unable to blink*

General diagnosis — Aplasia of facial, abducens, hypoglossal nerves, agenesis dig II V sin and dig II dex, syndactylia dig II—III dex, bronchopneumonia, hearing impairment?

Original hearing test — At 4 months hearing normal
Follow-up test — At $3\frac{3}{12}$ years, play audiogram hearing normal } Cf, fig 9, p 12

Comments — Since the patient was unable to blink the APR could not be used for the hearing test, the hearing could therefore be assessed only by the waking tests The original finding of non functioning upper branch of the facial nerve was confirmed by electromyography

CASE 16 E L ♂ b 25 4 57

Perinatal status — Cranial trauma at 7 months, lost consciousness, with spasms, gradual improvement, but paralysis of right facial nerve persisted

General diagnosis — *Subarachnoid haemorrhage, paralysis of right facial nerve,* cystopyelitis, rickets, secondary anaemia, hearing impairment?

Original hearing test — At 11 months hearing normal
Follow up test — At 4 years, play audiogram hearing normal } Cf, fig 9, p 12

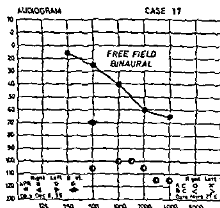
CASE 17 W E ♀ b 2 1 55

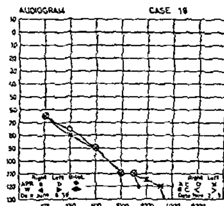
Pregnancy — Several minor haemorrhages during the last 3 months

Delivery — Two months premature, weight at birth 1510 g

Peri natal status — Attacks of cyanosis during first week

General diagnosis — Cerebral paralysis hearing impairment?





APR negative waking test negative
June 18 56

Original hearing test — At 12 months at 500 cps, hearing normal for both ears, at other frequencies, bilateral hearing loss with recruitment

Follow-up test — At $6\frac{3}{12}$ years, play audiogram findings as for original test

Comments — At 12 months the patient could be wakened only at 500 cps and 70 dB, but not at other frequencies even if the sound intensity was increased by 15 dB The APR was elicited at all frequencies According to the chart in Fig 8 hearing was normal at 500 cps and there must have been hearing impairment with recruitment at other frequencies, this was confirmed by the play audiograms at $6\frac{3}{12}$ years On this occasion the APR was elicited at all frequencies at normal level, so that the diagnosis of recruitment was also correct

CASE 18 A.B ♀ b 26 6 55

Pregnancy — Repeated spasm attacks

General diagnosis — Hearing impairment?

Original hearing test — At 12 months severe bilateral hearing impairment

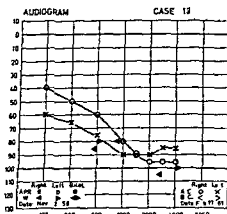
Follow-up test — At $4\frac{4}{12}$ years, play audiogram severe bilateral hearing impairment

CASE 19 M L ♀ b 8 8 55

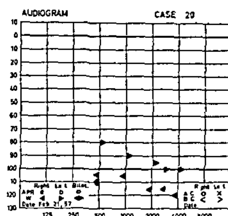
Heredity — Parents and sister deaf

General diagnosis — Hereditary deafness?

Original hearing test — At 16 months bilateral hearing impairment with hearing over whole frequency range



APR negative Nov 12, 56



APR positive only at right ear at 500 c/s Feb 21, 57

Follow-up test — At $4\frac{1}{12}$ years, play audiogram severe bilateral hearing impairment *with hearing over whole frequency range*

Comments — Waking test at 16 months gave the impression that the hearing impairment was not so severe as the play audiogram subsequently showed it to be It is therefore possible that there was a deterioration after the first measurement, as is usually the case in hereditary deafness, and as was noted for her elder sister, whose hearing worsened considerably over the last 5 years

CASE 20 J E ♀ b 27 9 55

Delivery — Twins, the patient being the last to be born

Peri natal status — Delayed development

General diagnosis — Oligophrenia, hearing impairment?

Original hearing tests — At 17 months bilateral hearing impairment with residual hearing

Follow-up test — At $5\frac{8}{12}$ years, informal tests, verification uncertain

Comments — Low intelligence ruled out play audiogram The patient definitely heard tones of high intensity APR negative

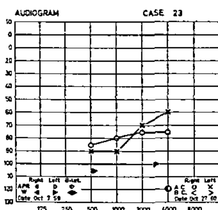
CASE 21 R E ♀ b 27 6 55

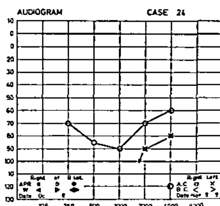
Peri natal status — Frequent spasm attacks, lost consciousness

General diagnosis — Oligophrenia, hearing impairment?

Original hearing tests — At 18 months severe bilateral hearing impairment (APR negative, waking test negative)

Follow-up test — Patient died at $2\frac{1}{2}$ years





CASE 22 S A ♂ b 6 5 55

General diagnosis — Progressive encephalopathy, hearing impairment?

Original hearing test — At 22 months hearing normal (Cf, fig 9 p 12)

Follow up test — At $5\frac{11}{12}$ years informal tests hearing normal

Comments — Attacks of spasms with loss of consciousness at 12 months, the disease is progressing

CASE 23 S.L. ♀ b 17 6 57

Heredity, pregnancy, delivery and peri natal status unknown adopted child

General diagnosis — Hearing impairment?

Original hearing test — At 16 months severe bilateral hearing impairment with best residual hearing in the higher frequency range for the left ear

Follow-up test — At $3\frac{4}{12}$ years, play audiogram as for original test

Comments — The type of hearing damage suggests intra uterine injury due to German measles

CASE 24 T C ♂ b 23 12 56

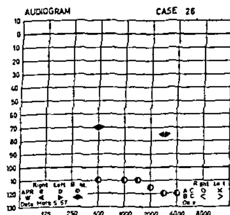
Pregnancy — German measles in the third month

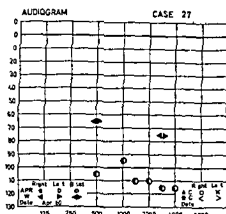
General diagnosis — Hearing impairment?

Original hearing tests — At 22 months severe bilateral hearing impairment Hearing recorded only at 4000 cps

Follow-up test — At $2\frac{11}{12}$ years play audiogram. hearing best at 4000 cps

Comments — The play audiogram showed the greatest hearing residue at 4000 cps The audiogram suggested intra uterine injury from German measles





CASE 25 H P ♂ b 21 12 56

Delivery — Breech presentation

Peri-natal status — Immediate post-natal period not remarkable Development thereafter delayed

General diagnosis — Mental debility, hearing impairment?

Original hearing test — At 18 months hearing normal

Follow-up test — At $4\frac{2}{12}$ years, play audiogram hearing normal

} Cf, fig 9 p 12

CASE 26 H.I ♂ b 24 9 54

Heredity — Child of mother's cousin born with eye deformity

Perinatal status — Both eye-balls absent at birth

General diagnosis — Oligophrenia, amaurosis, hearing impairment?

Original hearing test — At $2\frac{5}{12}$ years hearing normal

Follow-up test — At 6 years, informal tests hearing normal

Comments — No play audiogram recorded owing to low intelligence

CASE 27 K T ♂ b 20 9 57

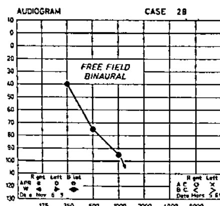
Delivery — Difficult Four days of labour, amniotic fluid passed 3 days prior to delivery

Peri-natal status — Spasms, late development

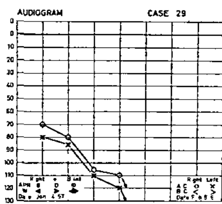
General diagnosis — Oligophrenia, hearing impairment?

Original hearing test — At $2\frac{8}{12}$ years, hearing normal

Follow-up test — At 4 years, informal tests hearing normal



APR and waking test 500—4000 c/s negative



APR and waking test 500—4000 c/s negative

Comments — On the first occasion waking tests were unsuccessful owing to too great depth of sleep as a result of hypnotics On this occasion the APR reflex was absent on both sides On the next occasion, in April, 1960, the test was carried out without previous administration of hypnotics Play audiogram not recordable owing to low intelligence

CASE 28 A M ♂ b 29 7 57

Pregnancy — Virus infection during first month

General diagnosis — Oligophrenia, epilepsy, hearing impairment?

Original hearing test — At $2\frac{4}{12}$ years severe bilateral hearing impairment

Follow up test — At $3\frac{7}{12}$ years, play audiogram findings as at original test

Comments — Delayed static development, low intelligence, I Q = 77

CASE 29 S R ♂ b 1 3 54

Delivery — Five days, amniotic fluid passed first day

General diagrosis — Hearing impairment?

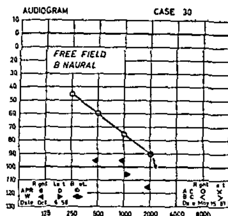
Original hearing test — At $2\frac{11}{12}$ years very severe bilateral hearing impairment, possibly totally deaf

Follow-up test — At $6\frac{11}{12}$ years, play audiogram findings as at original test

CASE 30 J K ♀ b 13 5 54

Delivery — Ten weeks premature, weight at birth 1130 g

Peri natal status — Placed in incubator and given oxygen, weight fell to 880 g Parathypus while in incubator



APR negative bilat, waking test negative above 2000 c/s

General status — Mental debility, hearing impairment?

Original hearing test — At $4\frac{5}{12}$ years severe bilateral hearing impairment with residual hearing of use in training, possibly greater residual hearing in right ear

Follow-up test — At 7 years, play audiogram severe bilateral hearing impairment

Comments — The static development of the child was delayed, could not sit withouth support until 3 years of age, spastic, used mouth instead of hands Auditory training has given satisfactory results At 6 years understood 30 words, could speak some words, was lively, gay and active

Owing to her spasticity there was great difficulty in recording a play audiogram, which was therefore made by the free-field technique

Summary of results

The follow-up examination of the 10 normal subjects showed that all had normal hearing except one In this case the audiogram displayed a hearing loss in one ear amounting to 20 dB in the range 125—500 cps This proved to have been due to recent otitis

This part of the study thus showed that the follow up play audiograms confirmed the results of the original APR and waking tests

In distinction to the normal group, where the preliminary hearing test was performed during the first day of life, the children in this group with suspected hearing impairment had at the preliminary test been of different ages The medical diagnosis was highly variable, but most of the patients were classed as cases of neonatal haemolytic disease with jaundice Four of the children have died and 7 displayed very low intelligence

The group with suspected hearing impairment consisted originally of 30 cases, 4 of whom died before the control examination of the hearing could be made (Cases 3, 13, 14, 21, Table 2) Of the remaining 26 cases, 20 were followed up with play audiometry while 6 had such low intelligence that informal hearing tests had to be resorted to

In all but 2 of these 26 cases the results of the original hearing tests were verified by one of these two hearing tests

In one of the exceptions (case 6) in which the hearing was judged to be normal on the third day of life but impaired at $2\frac{5}{12}$ years, it appeared that the preliminary findings were correct but that there was a severe reduction—probably of genetic origin—later on

In the second exception (case 20) the intelligence was so low that the control examination neither contradicted nor confirmed the preliminary findings

Table 2 Group with suspected hearing impairment

Case	Sex	General Diagnosis	Original Hearing Test			Follow up Test			Comments
			Age	Hearing Normal	Hearing Loss	Age	Confirmation by audiogram	Confirmation probable	
1 G P	F	Asphyxia neonatorum c atelectas *pulm* immaturitas + cataract + vitium org cord cong + haemorrhagia intracran + surditas?	< 1 month	+		4 y 9 m		+	
2 P S	M	Dysontogenesis auris sin et mandibulae sin + surditas?	»	right	left	4 y 10 m	+		
3 U M	F	Mikrocephalia + surditas?	»	»					Died at *7 months*
4 W O	M	Surditas hereditaria?	»		+	3 y	+		
5 R	F	» »	»	+		4 y	+		
6 H A	F	» »	»	+		2 y 5 m	not verified		
7 L	M	» »	»	+		3 y 10 m	+		
8 J T	M	Morbus haemolyticus *neonatorum* + icterus nucleosus + surditas?	»	+		3 y 4 m	+		
9 B	F	Morbus haemolyticus neonatorum + surditas?	»	+		3 y 5 m	+		
10 J A	M	Surditas?	1—3 months	+		4 y 5 m	+		
11 O	M	Morbus haemolyticus neonatorum (A immunisation) + hydrocephalus incertae causae + surditas?	»	+		4 y 9 m	+		
12 L	M	Immaturitas + encephalopatia + surditas?	»	|		4 y		+	
13 P A	F	Status post morbus haemolyticus neonatorum + icterus nucleosus + bronchopneumonia + surditas?	3—12 months		+				Died at 7 months
14 S	M	Encephalopatia + status post osteomyelit + amaurosis congenita bilat + surditas?	»	+					Died at 2 years

Case	Sex	General Diagnosis	Original Hearing Test			Follow up Test			Comments
			Age	Hearing Normal	Hearing Loss	Age	Confirmation by aud ogram	Confirmation probable	
15 W E	F	Aplasia nuclei facialis — abduens — hypoglossus + agenesia dig II + V sin et dig II dx + syndactylia dig II—III dx + bronchopneumonia + surditas?	»	+		3 y 3 m	+		
16 E L	M	Haemorrhagia subarachmoidalis + paresis nervi fac dx + cystopyelit + rachitis + anaemia sec + surditas?	»	+		4 y	+		
17 W E	F	Paralysis cerebralis + surditas?	1—2 years		+	6 y 3 m	+		
18 A B	F	Surditas?	»		+	4 y 4 m	+		
19 M L	F	Surditas hereditaria?	»		+	4 y 1 m	+		
20 J E	F	Oligophrenia + surditas?	»		+	5 y 8 m		uncertain	
21 R E	I	» »	»		+	—			Died at 2 ½ y
22 S A	M	Encephalopatia progressiva + surditas?	»	+		5 y 11 m		+	
23 S L	F	Surditas?	»		+	3 y 4 m	+		
24 T C	M	Surditas?	»		+	2 y 11 m	+		
25 H P	M	Debilitas psychica + surditas?	»	+		4 y 2 m	+		
26 H I	M	Oligophrenia + amaurosis + surditas?	> 2 years	+		6 y		+	
27 K T	M	Oligophrenia + surditas?	»	+		4 y		+	
28 A M	M	Oligophrenia + epilepsia + surditas?	»		+	3 y 7 m	+		
29 S R	M	Surditas?	»		+	6 y 11 m	+		
30 J K	F	Debilitas psychica + surditas?	»		+	7 y	+		
Total 30				18	13		19	5	4 died

Table 2 Group with suspected hearing impairment

Case	Sex	General Diagnosis	Original Hearing Test			Follow up Test			Comments
			Age	Hearing Normal	Hearing Loss	Age	Confirmation by audiogram	Confirmation probable	
1 G P	F	Asphyxia neonatorum c athlectas pulm, immaturitas + cataract + vitium org cord cong + haemorrhagia intracran + surditas?	< 1 month	+		4 y 9 m		+	
2 P S	M	Dysontogenesis auris sin et mandibulae sin + surditas?	»	right	left	4 y 10 m	+		
3 U M	F	Mikrocephalia + surditas?	»	»					Died at 7 months
4 W O	M	Surditas hereditaria?	»		+	3 y	+		
5 R	F	» »	»	+		4 y	+		
6 H A	F	» »	»	+		2 y 5 m	not verified		
7 L	M	» »	»	+		3 y 10 m	+		
8 J T	M	Morbus haemolyticus neonatorum + icterus nucleosus + surditas?	»	+		3 y 4 m	+		
9 B	F	Morbus haemolyticus neonatorum + surditas?	»	+		3 y 5 m	+		
10 J A	M	Surditas?	1—3 months	+		4 y 5 m	+		
11 O	M	Morbus haemolyticus neonatorum (A immunisation) + hydrocephalus incertae causae + surditas?	»	+		4 y 9 m	+		
12 L	M	Immaturitas + encephalopatia + surditas?	»	+		4 y		+	
13 P A	F	Status post morbus haemolyticus neonatorum + icterus nucleosus + bronchopneumonia + surditas?	3—12 months		+				Died at 7 months
14 S	M	Encephalopatia + status post osteomyelit + amaurosis congenita bilat + surditas?	»	+					Died at 2 years

Summary

For measuring the hearing of children less than 3 years old it is necessary to use objective methods This is true also of older children who for some reason are unable to cooperate in play audiometry, (say because of intelligence defect, immaturity, or spasticity) In both kinds of subjects, I have performed two kinds of objective hearing tests

I Threshold determination of the auropalpebral reflex (APR) consisting in the contraction of the orbicularis oculi muscle

II Determination of the intensity of sounds required to waken the cihld who is in a certain depth of sleep A specially designed tone audiometer was used for these tests

Those tests are conducted with the child lying on its side in a cot In the first 20 subjects, who had been selected as probably being normals, the APR was elicited at a threshold of 105—115 dB for all the frequencies tested in the range 500—4000 cps

That it is possible to use these threshold determinations of APR as a test of hearing level in the *newborn* is due to the similarity between APR and another acoustic reflex, the stapedius reflex. this latter function is familiar in both normals and persons with defective hearing The APR reflex-threshold curve for *adults with normal hearing* are very similar, also from the anatomic aspect the two reflexes have much in common The afferent part (acoustic nerve) and the efferent part (facial nerve) of the reflex are common to both but the centre of the stapedius reflex is situated in the pons and that of the APR in the reticular formation

The APR threshold curve for the children examined should indicate normal hearing In the individual case, however, the fact that the APR has been elicited at a particular frequency with a tone of "normal" intensity cannot be taken as proof that the hearing for the frequency in question is normal As has been shown in the case of the stapedius reflex, there may be severe hearing loss with recruitment So as to distinguish between normal hearing and impaired hearing with recruitment, experiments were performed to find the intensity required to awaken a child in a depth of sleep such that the APR could be elicited by tactile stimulus The audiometer was the same as that used in the APR tests The frequencies were 500 and 3000 cps, and the tones were of 1—5

Discussion

It is evident from the results that the APR and waking tests for children are extremely reliable and objective, irrespective of the age at which they are performed and of the underlying cause of their disease. Whether they are performed during the first day of life or in the fifth year is of no consequence as far as the results are concerned. However, the younger the child is the easier the tests are to perform. A newborn is extremely easy to handle; only half a minute or so after a waking test the infant will have fallen asleep again, so that the test can be repeated. In the case of the older child, however, the examiner may have to wait several hours for the child to fall asleep again.

In order to simplify the time-consuming waking test in the case of the older children note was taken of the change in breathing rhythm that precedes waking. Just before the child opened its eyes the respiration changes, as a result of the acoustic stimulus, from a regular rhythm with moderate excursions of the chest to rapid irregular and deep movements. In no case were changes in respiration, due to acoustic stimulation when the child is in a deep sleep, observed. This would seem to be a fairly reliable sign of wakening and the stimulus can then be discountinued so that the child falls asleep again, the waking test can then be repeated on another frequency.

In the APR threshold determination, repeated stimulation leads to a tendency for habituation. The response may then be diminished or even abolished for the tone frequency under test. It is then necessary to try another frequency and then gradually return to the previous one, for which the reflex response will again be elicited. It was found that the older the child is the greater the risk of habituation, although this was not uncommon even among newborn. The determinations of the APR threshold therefore takes a much longer time for older children than for newborn. There is a large variation from one subject to another, however.

The procedure requires no cooperation at all on the part of the child, so that it is possible to test the hearing of children with very low intelligence quotients and in cases where other methods could not be used. An objective determination is made of the sound intensity required to attain a reflex threshold and to waken a child from a certain depth of sleep.

With these hearing tests it has been possible to determine with great confidence whether the hearing of a child (newborn or older) is normal or impaired, and to judge the type and degree of the impairment. In some cases (conductive and retrocochlear loss) it is also possible to record an audiogram. The procedures are of great value for testing the hearing where a play audiogram cannot be recorded — children less than $2\frac{1}{2}$ years, and older children who for some reason, such as low intelligence or spasticity, cannot cooperate in producing a play audiogram.

© ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta
OTO-LARYNGOLOGICA

S U P P L E M E N T U M 176

DEAFENING EFFECTS OF NOISE ON THE CAT

BY

JAMES D. MILLER, CHARLES S. WATSON,
AND WALTER P. COVELL

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 176

DEAFENING EFFECTS OF NOISE ON THE CAT

BY

JAMES D. MILLER, CHARLES S. WATSON, AND WALTER P. COVELL

From Central Institute for the Deaf, St. Louis, Missouri,
Hearing and Communication Laboratory, Indiana University, Bloomington, Indiana,
and Department of Otolaryngology, Washington University Medical School,
St. Louis, Missouri

Almqvist & Wiksells
BOKTRYCKERI AKTIEBOLAG
UPPSALA 1963

FOREWORD

The behavioral investigations reported in this monograph were conducted by Dr James D Miller and Mr Charles S Watson[1] at the Hearing and Communication Laboratory of Indiana University Professor James P Egan who is director of the laboratory was responsible for the initiation of the project and he was closely involved in its conduct Clint D Anderson Gordon Z Greenberg and Richard F Gundy assisted in several phases of the behavioral program

The histological studies were conducted by Dr Walter P Covell at the Department of Otolaryngology of Washington University Medical School in St Louis Missouri

The authors wish to acknowledge the value of conversations with Dr Donald H Eldredge of Central Institute for the Deaf concerning the planning of the exposures and the interpretations of the audiometric and histological findings We also wish to express our appreciation to Dr W D Neff of Bolt Beranek and Newman Inc for instruction in the surgical techniques for the destruction of cochlea and for many helpful suggestions concerning threshold testing as well as the care of the laboratory cat

The United States Air Force provided the major support for this research under Contracts AF 33(616) 3844 and AF 33(616) 3637 monitored by the Biomedical Laboratory Aerospace Medical Laboratory Wright Air Development Division Air Research and Development Command Wright Patterson Air Force Base Ohio Supplementary support was kindly provided to the Hearing and Communication Laboratory by the Deafness Research Foundation by the Allison Division of General Motors Indianapolis Indiana and by the Behavioral Sciences Division of the Air Force Office of Scientific Research Washington 25 D C Final stages of data analysis and preparation of the manuscript were completed after the senior author was appointed to the staff of Central Institute for the Deaf St Louis Missouri and the support of the Institute through grants from the National Institute of Neurological Diseases and Blindness is gratefully acknowledged

[1] Mr Watson is now at the Department of Psychology and the Defense Research Laboratory of the University of Texas

CONTENTS

I. INTRODUCTION

The relations between deafness and exposure to noise have been studied in clinical cases and by field survey methods (see, as examples, reports by Rudmose 1957, or Nixon and Glorig 1961). In the laboratory, histological and electro-physiological methods have been used most often (see, for example, Davis and Associates 1953). In addition, inferences about acoustic trauma and noise-induced deafness have been drawn from laboratory investigations of temporary threshold shifts (see, as examples, Ward, Glorig and Sklar 1958, 1959*a*). Another laboratory method, a method of behavioral audiometry with experimental animals, was used in the experiments to be reported in this monograph.

This method has been previously used to advantage by Lindquist, Neff and Schuknecht (1954), and it utilizes the following kinds of procedures. Laboratory cats are trained to respond to tones, and measures of their auditory sensitivity are made before and at intervals after an exposure to noise. These measures of auditory sensitivity and their time course over the post-exposure interval are used to define the following variables. A threshold shift is defined as the post-exposure threshold, expressed in decibels, minus the pre-exposure threshold. If a threshold shift is measured at a time that exceeds a few seconds after the cessation of the exposure and if this threshold shift declines to zero over time, the animal is said to have suffered a temporary threshold shift (TTS). If threshold shifts are measured that are stable and persist over a period of several weeks, then these threshold shifts are said to be persistent threshold shifts (PTSs) and permanent injury to the auditory mechanism is inferred. Threshold shifts that have both temporary and *fresh* persistent components are defined as compound threshold shifts (CTSs). (The compound threshold shift is a new term and it is discussed in Chapter VI.)

Although each of the methods mentioned above provides a particular kind of valuable information, the method of behavioral audiometry with laboratory animals has certain advantages for the investigation of noise-induced deafness and acoustic trauma. Precise control and specification of the conditions of the exposure are possible. Audiometric measures can be made on both before, during and after exposures to noise. In contrast to most electrophysiological and all histological methods, the methods of making auditory measurements do not disturb or interfere with the later performance of the auditory mechanism. A compelling advantage of a method of behavioral audiometry is the fact that changes in the audiogram clearly define one of the most important aspects of what is meant by damage or injury to *hearing*.

Of course, a particular method can only answer a limited range of questions. Certain functional and structural changes produced by noise can only be determined by physiological and anatomical methods. Inferences to the human species based on data from another species must be made with care. Practical disadvantages of behavioral audiometry with animals are the time-consuming tasks of training the animals and testing their auditory sensitivity.

Utilizing a method of behavioral audiometry, we collected data which are relevant to the following issues: the audibility curve of the cat, growth of persistent threshold shift (PTS) with increasing durations of exposure to noise, the effect of breaking up an injurious exposure into a series of short, spaced exposures to noise, the relation between TTS audiograms and PTS-audiograms, the relation between PTS audiograms and the spectrum of the noise, amount and period of recovery after exposure to the noise, and the relation between traumatic effects of noise in the human and the cat ear. In addition, a histological evaluation was made of each cat's cochlea; it is therefore possible to present an evaluation of the structural changes produced by the exposure as well as data on the relation between the behavioral and the histological findings.

II SUBJECTS AND AUDIOMETRIC PROCEDURES

Introduction

Cats were used as subjects and they are described below. The cats were trained to respond to a tonal stimulus by the method of instrumental avoidance conditioning. The learned response, after extended training and with special precautions, was used to determine behavioral thresholds in a psychophysical procedure. All training and testing were carried out in a proper acoustical environment. Although the essentials of the training and testing procedures have been previously described by Culler and Associates (1935) and by Neff (1947), the apparatus and methods used in the present experiments are described in detail in this chapter.

The cats

Forty-seven cats were used in these experiments. All were mongrels and all were estimated to be between one and four years of age. Forty-two were trained in order that their auditory thresholds could be measured behaviorally. *Five were not trained; these were used only for histological preparations.* Nine of the trained cats (Cats Nos. 3, 4, 5, 13, 19, 20, 23, 24 and 25) were used in preliminary experiments whose procedures differed slightly from those described in the present paper. The details of the preliminary experiments are given in a previous report by Miller and Associates (1958).

All of the cats were in excellent general health during the course of these experiments. This was largely due to a high quality diet and to a one- to two-month period of isolation of new cats. During this isolation period, sickly cats were eliminated, while the remainder showed the beneficial effects of the laboratory diet. Two specific selection procedures tended to eliminate cats with hearing disorders: (1) those cats that were difficult to train were discarded, and post mortem examination of these cats often revealed a middle ear infection, and (2) if an infected ear was encountered during surgery (see below), the cat was eliminated from the colony.

Aside from the exceptions noted in the text, only monaural cats were used in the behavioral experiments. These cats were prepared by surgical destruction of the left cochleas, and each operation was performed under sterile conditions while the cat was deeply anesthetized with sodium pentobarbital. Most often this operation was performed on a cat before his training was initiated, and since the operation was followed by a severe vestibular upset, it was necessary to wait several weeks after the operation before beginning

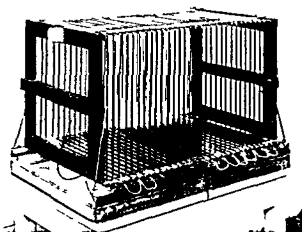


Fig 1 The double grill cage used for the training and testing of the cats

training or testing procedures Histological examination of the operated ears showed that the cochlea had been completely obliterated in all but two cases The upper turns of Cat 20 s left cochlea could be identified but none of the sensory cells appeared to be in functional condition In the case of Cat 71 a few inner hair cells in the upper turns appeared to be healthy and possibly functional

All but the nine cats used in the preliminary experiments were treated as a prophylactic measure for four to seven days after surgery with penicillin Similar prophylactic treatment with a dihydrostreptomycin penicillin mixture (Combiotic C Pfizer and Co) was given to the nine cats used in the preliminary experiments Even though dihydrostreptomycin may produce ototoxic effects none were detected in the present experiments that is the threshold and threshold shift data of the preliminary experiments are in close agreement with the remainder of the data

Apparatus

A double grill cage (Fig 1) was used for the training and testing of the cats In its interior dimensions this cage was 33 in long 18 in wide and 18 in high A hurdle 5 in high divided the cage into two compartments each was 16 5 in long The floor of the cage was a grid formed of lengths of rectangular brass stock (¼ in by ½ in) which were spaced center to center at 1 in intervals The top and two sides of the cage were grids formed by brass rods (¼ in diam) which were welded to the grid bars of the floor Wooden frames covered with hardware cloth were used as doors at the ends of the cage The unit was mounted on an iron base but was insulated from it by Lucite runners Lucite stringers along the top of the cage added strength to the construction The double grill cage could be electrified to act as an

aversive stimulus. The shock was controlled manually by means of a push button and by means of a rheostat available for intensity adjustment. The two compartments of the double grill cage were electrically independent, the compartment to be shocked being selected from the experimenter's console.

A buzzer was mounted on each of the doors of the double grill cage. These buzzers generated an overall SPL[1] of about 70 db in the cage. Also they provided, via the grid bars, a vibratory stimulus to the cat's feet. The sound of the buzzer served as a secondary aversive stimulus in the training procedure to be described.

For the hearing tests an audiometer assembled from high quality electronic components was used. An audio oscillator, an electronic switch, an amplifier, attenuators, and matching transformers were wired into switch panels so that circuits could be selected which were appropriate to the hearing level of the cat and the desired test frequency. The loudspeakers, the acoustic environment, and the calibration of the sound field are described in the next section of this chapter.

The auditory stimulus for a training or testing trial consisted of either a continuous or a pulsed tone which was followed by the sound of the buzzers. The durations of these events were controlled by a reliable multi-cam timer. A trial could be initiated by a momentary contact and it could be terminated at any time after its onset. The exact durations of the auditory stimuli are given in the descriptions of the training and testing procedures.

Acoustic Environment and Calibration of Audiometer

For the threshold tests the double grill cage was placed on a table in a sound insulated chamber of room-within-a-room construction. The outer room had a concrete floor, concrete block walls of 8 in. thickness, and a ceiling of preformed concrete slabs. This room's interior walls and ceiling were covered with 4 in. thick blankets of Fiberglas and its floor was carpeted. The inner room was an audiometric booth (Industrial Acoustics Company Inc., Model 402) whose interior dimensions were approximately 64 × 72 by 78 in. The walls of this booth were 4 in. thick. Between the control room and the interior of the audiometric booth the attenuation was estimated to be about 80 db for the mid frequency range. One-way observation windows allowed the experimenter in the control room to view the interior of the audiometric booth.

Test tones were generated in the booth (Fig. 2) by a pair of 10 in. loudspeakers (Altec Lansing Co., Model 756A). For tests at 32 kc an Ionovac (Electro Voice Inc., Model T-3500) was used. These speakers were mounted in a cabinet; the center of the cabinet was on a line with the center of the double grill cage at a horizontal distance of 42.5 in. and 25.5 in. above it. Thus, the speakers were about 49.5 in. from the double grill cage.

[1] Throughout this monograph the reference level of 2 × 10⁻⁴ microbar is used.



FIG 2 The experimental arrangement for the audiometric tests The picture on the right shows a tester observing a cat The picture on the left shows the interior of the audiometric booth

The audiometer s sound field was calibrated by the following procedure For a specified voltage across the loudspeakers, the SPL of each of the test frequencies was measured at 54 positions within the double grill cage These positions were uniformly distributed throughout the interior of the cage The sound level meter consisted of a condenser microphone (Bruel and Kjaer, Type 4111), an amplifier (Bruel and Kjaer, Type 2610), a variable filter set (Spencer Kennedy Laboratories Inc , Model 302), and a vacuum tube volt meter (Ballantine Laboratories, Inc , Model 300) The calibration of the microphone supplied by Bruel and Kjaer was used for all frequencies except 32 kc At 32 kc the microphone was calibrated by the substitution method against a microphone whose sensitivity at 32 kc had been determined at the Bio Medical Laboratory of Wright Air Development Division, Wright Patterson AFB

The variability of sound levels across the 54 positions of the microphone is shown in Table 1 The top row shows the frequencies of the test tone The entries in the next three rows are deviations in decibels of the indicated percentiles from the median The last row of table 1 shows the semi interquartile ranges of these measurements Examination of Table 1 shows that, while the range of levels was large, for most frequencies 50 % of the positions fell within ±3 db of the median position

The contribution of variability of the sound field to variability of the threshold shift measures was attenuated by two facts (1) each cat developed a fairly stereotyped behavior pattern within the double grill cage, and, thus, limited the spatial variability of the sound field to a smaller range, and (2) each measurement included several determinations of the threshold We believe that averaging procedures inherent in these measurements reduced the error due to spatial fluctuations in the sound field

TABLE 1. *Spatial variability of the audiometer's sound field at fifty-four positions within the double-grill cage.*

The top row shows the frequencies of the test tone The entries in the next three rows are deviations in decibels of the indicated percentiles from the median The last row shows the semi interquartile range

Percentiles	Frequency in kc/sec 0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0
100th	4.7	6.5	6.0	5.0	9.5	8.0	5.5	7.7	6.0
75th	1.7	1.5	2.5	1.5	5.5	4.0	2.0	1.7	1.0
25th	−1.8	−1.5	−3.5	−3.5	−5.0	−5.5	−2.5	−1.3	−3.0
0th	−4.3	−5.5	−12.0	−12.5	−23.5	−23.5	−17.0	−18.3	−8.0
$\frac{75th-25th}{2}$	1.75	1.5	3.0	2.5	5.25	4.75	2.25	3.0	2.00

For calculation of the absolute values of the thresholds, it was decided to use the median SPL for each test frequency. While it is true that the median may not have been the best measure of the sound field for a particular cat, it was assumed that the typical cat would be sampling the typical value of the sound field

During the normal use of the audiometer the intensity of the signal was attenuated after an amplifier in the circuit and in this way the signal-to-noise ratio at the loudspeaker was maintained For a "high-power" arrangement of the audiometer, the signal was attenuated prior to amplification, and the output of the amplifier was placed directly across the loudspeakers Table 2 shows the median level of the sound field at the maximum output of the audiometer for both the normal and the high-power (High-P) arrangements of the circuit Also shown in Table 2 are the maximum threshold shifts that could be measured for an average, normal cat with either the normal or high-power circuits

TABLE 2 *Median SPL at maximum output for two arrangements of the audiometer circuit.*

Also shown are the maximum threshold shifts that are measurable for an average normal cat with the two circuit arrangements See text for explanation

	Frequency in kc/sec 0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0
Normal SPL	77.1	73.9	76.9	78.4	71.9	66.7	62.9	52.5	79.5
TS	[illegible]	58.9	72.9	88.4	88.4	79.7	80.4	63.0	73.0
High P SPL	[illegible]	100.9	102.4	103.9	97.9	91.9	86.9	78.0	—
TS	[illegible]	[illegible]	98.4	113.9	114.4	101.7	101.4	86.5	—

Training Procedure

A standard training procedure which evolved in the course of these experiments is described below

Stage 1 *Escape from shock training* Five to twenty trials of escape from shock training were given before the avoidance procedure was initiated Only five trials were given in a session, and a three minute inter trial interval was used On each trial the shock was gradually increased from an initial low level until the cat crossed the hurdle (Mean time for stage 1, 15 min, mean number of trials, 5)

Stage 2 *Avoidance training* The avoidance stimulus was a 2 kc tone presented at an SPL of about 60 db for 1 5 to 2 sec If the cat did not make the desired response during this period, then contiguous with the cessation of the tone was the onset of a buzzer whose duration was 1 sec Also, if the cat failed to cross the hurdle before the onset of the buzzer, electric shocks were applied intermittently until the escape response had occurred On the other hand, if after the onset of the tone the cat crossed the hurdle before the onset of the buzzer, the tone was immediately terminated and both shock and buzzer were avoided Thus, each training trial terminated with the response of crossing the hurdle For this training the inter trial interval was 2 min and no more than ten trials were given in a session (Mean time for stage 2, 2½ hr, mean number of trials, 75)

Stage 3 *Generalization training* After a cat reached a level of approximately 85 % avoidance responding in stage 2, stage 3 was initiated In this stage, by gradually shifting to frequencies above and below 2 kc, the cat was trained to respond at all frequencies that were to be used in the threshold tests Also, the inter trial interval was changed to a random schedule These intervals ranged from 15 to 90 sec, and the mean interval was 45 sec (Mean time for stage 3, 1 hr, mean number of trials, 80)

Stage 4*a* *Training in threshold procedure* A threshold procedure was introduced and used nearly as described below Often it was necessary to give extra trials at some intensities to train the desired behavior of responding to weak tones (Mean time for stage 4*a*, 6 hr)

Stage 4*b* *Stabilization of performance* Even after the cat had learned to respond to weak tones, additional practice in the threshold procedure was required in order to stabilize the performance (Mean time for stage 4*b*, 9 hr) The total time to train an average cat was 19 hr

Threshold Procedures

For the threshold tests the avoidance procedure was continued The parameters however, were varied as described below

The auditory stimulus consisted of a sequence of five tonal pulses Each pulse was 1 sec in duration, the silent interval between successive pulses was 0 5 sec The rise–fall time of a pulse was 0 1 sec The buzzer was sounded for 1 sec at the termination of the fifth tonal pulse

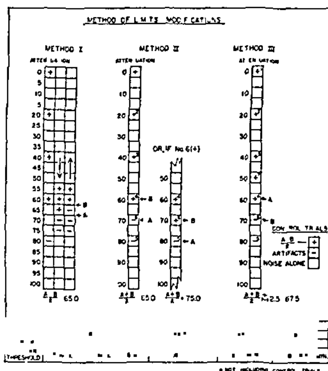


Fig 3 Sample record sheets which illustrate three psychophysical methods used for the determination of the cat's auditory sensitivity Decibels of attenuation re an arbitrary level are shown on the left hand side of each sample record The entry of a plus on the record indicates a successful avoidance response, a minus indicates a failure to respond to the tone The superscripts associated with the pluses and minuses indicate the ordinal position of the trial within the sequence The method used to calculate the threshold is illustrated for each method The several schedules of inter trial intervals that were tried with these methods are indicated along the bottom of the figure

In the determination of a *threshold*, a trial could terminate in three ways (1) the cat crossed the hurdle before the buzzer sounded, (2) the cat failed to respond before the buzzer, but did cross the hurdle after the onset of the buzzer (sometimes with the additional goad of shocks), or (3) the cat failed to cross the hurdle Throughout the test only crossings of the hurdle during the interval between the onset of the tone and the onset of the buzzer were considered successful avoidance responses, all other possibilities were referred to as failures to respond (to the tone)

The decision to shock an animal for failure to respond was based on the animal's previous behavior A cat was almost always shocked for failures to respond when the intensity of the tone was well above the previous value of his threshold At lower levels of the tone the experimenter shocked the animal for failures only when it appeared that the cat's performance was faltering in the direction of no responding When shock was given for failure to respond to the tone at a level near threshold the shock was usually mild It is important to remember that the buzzer sounded after every failure to respond

Three psychophysical methods that were used are illustrated by the sample records (Fig. 3). Decibels of attenuation re an arbitrary level are shown on the left hand side of each sample record. The entry in the record of a plus indicates a successful avoidance response, a minus indicates a failure to respond.

The schedules of inter-trial intervals (ITI) that were tried with these methods are indicated at the bottom of the figure. In each case the ITI was varied randomly within the indicated limits. The lowest line shows the mean time required for a single determination of a threshold. The several conditions for the inter-trial intervals shown for each method reflect our discovery that satisfactory results could be obtained with shorter intervals than we originally believed.

First consider Method I. Initially, an approximate threshold was determined using a descending series with a step interval of 20 db. This series is shown by the pluses and the minus in the left hand column of the example under Method I. About 20 db above the estimated threshold, a second descending series was begun. This descending series had a step interval of 5 db and it was continued until two successive failures were recorded. An ascending series was begun at the level of the last failure and continued until two successive pluses were recorded.

The descending threshold was defined as the level one half way between the level at which the last avoidance response had occurred and the level at which the first of the two successive failures had occurred. The ascending threshold was defined in an analogous manner. The threshold recorded for the measurement was the average of the ascending and descending thresholds. This scoring procedure is indicated in Fig. 3.

It was noticed that the approximate threshold determined as the first step in Method I was usually very close to the threshold arrived at by the full procedure. This fact suggested Method II which is also illustrated in Fig. 3. Method II is a simple descending series with a step interval of 20 db. In addition, after a failure to respond had occurred, the intensity of the tone was raised 10 db and another trial was run. The threshold was taken as the level one half way between the lowest plus and the highest minus.

Methods III*a* and III were complications of Method II. They started as a descending series with a step interval of 20 db. However, after the first failure was encountered, a bracketing procedure was introduced.

A sample record sheet for Methods III*a* and III is shown in Fig. 3 and a detailed description of these methods is as follows:

(1) On the first trial the tone was presented 40 to 60 db above threshold.

(2) After a plus trial, the intensity of the tone was decreased 20 db.

(3) After a minus trial that was immediately preceded by a plus trial, the intensity of the tone was increased 10 db.

(4) After a minus trial that was immediately preceded by a minus trial, the intensity of the tone was increased by 20 db.

(5) An approximate threshold was calculated when the following three

conditions were met (*a*) pluses were recorded at two intensities separated by 10 db (*b*) minuses were recorded at two intensities separated by 10 db and (*c*) trials had been run at all 10 db steps between the pair of adjacent pluses and the pair of adjacent minuses

These five steps constituted Method III*a* For Method III the following additional steps were included

(6) Three additional trials were used to complete the measurement as follows (*a*) *Tone plus artifacts and noise* This was a trial given with the level of tone at the approximate threshold If the cat responded at this level 2.5 db of attenuation was added to the approximate threshold and if he failed to respond 2.5 db was subtracted (*b*) *Artifacts plus noise* This trial was run exactly as trial (*a*) however a resistor was substituted for the audio oscillator (*c*) *Noise alone* With the equipment set as described for (*a*) an interval equal to the duration of the tone was timed with a stop watch and the occurrence or non occurrence of a response was noted The order of the trials described under (*a*) (*b*) and (*c*) was randomized While the trials described under (*b*) and (*c*) did not enter into the calculation of the threshold a sufficient number of such trials provided the following additional information (1) the false alarm rate at threshold and (2) the response rate in the presence of possible artifacts

(7) Occasionally thresholds were obtained which deviated widely from the previous result on the same cat To reduce the probability of such a measure influencing the data any threshold which deviated by more than 20 db from the previous result was rejected In this case the measurement was immediately repeated this second measurement was *always* accepted

Experimental Comparison of Audiometric Methods I, II, and III a

A series of observations was made to compare Methods I II and III*a* For this comparison the mean inter trial interval was 46.5 sec for all methods All measures were accepted Method III*a* was used as described in steps 1 to 5 above For this procedure two test sessions were run using each method for each of 17 cats A test session consisted of threshold measurements at each of the frequencies at the nine one octave steps from 0.125 kc to 32 kc The order of test frequencies was randomized within each test session

As shown (Fig 4) the mean thresholds obtained by these three methods differed by less than one decibel Since all three methods gave the same absolute values for the thresholds they could only be distinguished on the basis of reliability time and suitability for long term testing of the cats A comparison of the methods with respect to reliability and time is shown in Fig 5 The ordinates are measures of within subject variability while the abscissas are the times required to obtain the data For each method the points from left to right are the estimated standard errors of the means based on 1 2 [illegible] observations respectively These standard errors were estimated using the data for all frequencies It can be seen that for a given level of

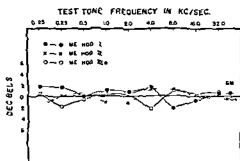


Fig 4

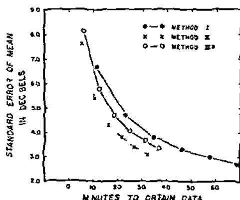


Fig 5

Fig 4 A comparison of mean thresholds obtained using Methods I II and III*a* The ordinate for each point is the deviation in decibels of the mean obtained by a particular method from the mean for all three methods Similar results are shown for the grand means over all frequencies

Fig 5 A comparison of the intra-cat reliability for Methods I II and III*a* based on the standard error of the mean threshold (σ_R) using 1 to 6 observations The ordinates are standard errors of the means in decibels while the abscissas are times in minutes necessary to obtain the required number of measurements For each method the data points from left to right show the expected standard error of the mean threshold for 1 2 and 6 determinations respectively For example *the fourth open circle from the left shows that four measurements using Method IIIa would* require about 24 8 min and the standard error would be about 4 05 db

reliability Method I requires fewer threshold determinations but more time than Methods II or III*a*

Indeed since Method II gave the greatest reliability for the least expenditure of time it would appear to be the method of choice Method III was finally selected however because it provided more opportunities than Method II for animals to respond to the very weak tones and, thus it was thought to be more suitable for the long term testing of the cats

The schedule of inter trial intervals was shortened to a mean value of 28 5 sec with a range of 15 to 50 sec In this form Method III required about 5 min per threshold While the variability of thresholds collected using Method III in its complete form is given in detail in Chapter III, it may be mentioned that Method III in its final form was more reliable and faster than Methods I II or III*a* the standard error of a single measurement being about 6 15 db

III MINIMUM AUDIBLE FIELD OF CAT

Introduction

Behavioral measurements of the cat's audibility curve have been more extensive than those for any other sub-human species. Dworkin and Associates (1940) published the first extensive set of data on the cat's audibility curve. These authors, however, did not calibrate their sound field by direct measurement. Kryter and Ades (1943) published thresholds at three frequencies for which the sound field had been calibrated. Neff and Hind (1955) published an audibility curve for the cat based on direct calibration of the sound field for frequencies at and below 16 kc. These authors also showed that the cat could hear at frequencies as high as 60 kc. Recently McGill (1959), Elliott, Stein and Harrison (1960) and Miller and Associates (1958) have also measured audibility curves for the cat in calibrated sound fields.

During the course of the present experiments audibility curves were obtained under standard conditions for each of thirty-four highly trained cats. These cats were tested using Method III, which is described in the previous chapter. The test frequencies were the nine one-octave steps from 125 to 32 000 cps. A cat was tested at all nine frequencies in each of five sessions, and the order of frequencies was randomized within each session. At each test frequency a cat's threshold was defined as the mean of the five measurements. These means served as the basis for the calculation of threshold shifts in the deafness experiments, and they are presented in this chapter as a contribution to our knowledge of the cat's auditory sensitivity.

Variability of Thresholds

Fig. 6 shows the variability among cats. At most frequencies 50 % of the cats fall within ±3.5 db of the median cat; furthermore, almost all of the data are included within ±15 db of the median. Only Cat 84 consistently deviated from the median by more than 20 db. This cat was dropped from the experiments, and a post mortem examination showed that cerumen was impacted in the ear canal in the region of the drum. The overall agreement among the cats becomes even more striking when the spatial variability of the sound field is recalled (see Table I). Certainly some of the variability among cats can be accounted for by the variability in the sound field.

The within-cat variability was estimated using all data, both pre- and post-exposure, collected in these experiments. For this purpose approximately 627 threshold measurements (19 thresholds for each of 33 cats) were available for each of the nine frequencies. Fig. 7 shows the relation between the estimated [illegible] standard deviation (σ) and frequency of test tone. This

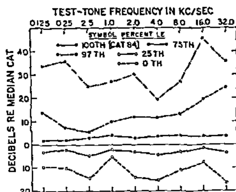


Fig 6

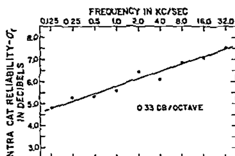


Fig 7

Fig 6 Variability between cats in their quiet absolute thresholds The frequency of the test tone is shown along the top of the figure, and the deviation of the indicated percentile from the median is shown on the axis of ordinates Cat 84 was not used in the trauma experiments

Fig 7 Intra-cat reliability for the nine frequencies of the test tone At each frequency, each cat s variance was calculated The measure of reliability shown on the axis of ordinates is the square root of the mean of the estimated population variances of all cats used in these experiments

standard deviation increases about 0 33 db for every octave increase in frequency For practical purposes, however, one estimate of the intra cat variance was used for frequencies at and below 1 0 kc and another was used for frequencies at and above 2 0 kc The square roots of the average values of σ_r^2 were 5 24 and 6 80 for the low and high frequencies, respectively To estimate the reliability of a mean, that is, its standard error for the particular sample of cats (σ_R), the formula is

$$\sigma_R = \frac{\sigma_r}{\sqrt{(Nn)}},$$

where N is the number of cats and n is the number of threshold determinations per cat The number obtained by this calculation is probably an under estimate of the true σ_R, since each estimate of this measure of reliability is based on the variability of the threshold over a period of 3 or 5 *successive* days The variability for measures separated by longer periods, say several days weeks, or months, is undoubtedly greater This means that if a cat s threshold were measured at 1 0 kc on 5 successive days and these tests were repeated one month later, a test for the significance of the difference between the means of each group of 5 measures would be biased toward a significant result This bias is noted here, since this type of statistical test is used in later chapters of this monograph

False-Alarm Rates and Artifacts

Recent investigations have demonstrated the important influence of procedural variables on detection behavior (see, for example, Swets, 1961) In addition on both procedural and theoretical levels these investigations have suggested that a threshold should be thought of as equivalent to an

index of sensitivity and that any additional connotation is questionable. The hit rate (response rate to tones) in a simple yes-no experiment is dependent on the false alarm rate (rate of spontaneous responses) as described by an operating characteristic. Now, even though the procedures of the present experiment are much more complicated than the simple yes-no experiment, the cat's hit rate is undoubtedly dependent on his false alarm rate and thus, his threshold values will be dependent in some complex way on his false alarm rate. Moreover, it is likely that an animal's false alarm rate is more closely related to the immediate experimental context than is the false alarm rate of the human listener. For, while the animal's behavior is oriented to the avoidance of shock, the human listener's behavior is usually oriented by the verbal concept of "hearing" given in the instructions. Thus it may be more important to control and describe the hit and false alarm rates in the case of sub-human listeners than it is in the case of human listeners.

When the hit and false alarm rates are specified at threshold values of the signal, in a sense, the cat's criterion is described. The procedures of the present experiment (see Method III, step 6 in Chapter II) represent a move in the direction of better control and description of these response rates and in this spirit the following data were collected. The proportion of responses to tones presented at the approximate threshold was 0.485. The proportion of responses to possible artifacts was 0.038. And the proportion of responses on noise-alone trials was 0.044. Each of these proportions was based on 1530 trials.

Under the assumption that these data could be treated as describing a point on an operating characteristic, the threshold levels given in this paper can be interpreted as levels which have a detectability index, d_s, of about 1.7. A clear explanation and definition of this index is given by Egan, Greenberg and Schulman (1961). The advantage of describing the threshold value in terms of a constant detectability index is that this number may be relatively independent of procedural variations.

Since the cats in our experiments could detect a tone generated by a 10 microvolt potential across the loudspeakers' voice coils, the possibility of artifacts of similar magnitude could not be ignored. Although artifacts were not audible to human listeners, it was still possible that the cat could hear them. The control apparatus for our audiometer, in fact, did produce clicks of very small magnitude in the audio circuits, and while these clicks could be observed on an oscilloscope, we were not able to measure them. In general, it should be remembered that artifacts are always associated with a tone presentation and that their elimination is a matter of degree. In relation to this problem, the closeness of the response rate to artifacts (0.038) to the rate on noise-alone trials (0.044) was an important finding, since it proved that the cats did not respond to artifacts when these artifacts were presented in isolation. Also, the audibility curves show that artifacts did not control the cat's response. Our experiments, however, do not eliminate the possibility that under some conditions artifacts could serve the function of a ready signal.

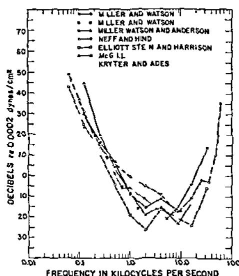


Fig 8

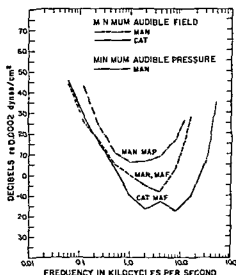


Fig 9

Fig 8 Audibility curves of cat Median results are shown for five previous sets of data from calibrated sound fields and for two new sets of data (filled circles and squares) The values of the median thresholds are shown in decibels re 0 0002 dynes/cm² along the axis of ordinates, and the abscissas are frequencies of the test tones shown in kilocycles per second The test tone frequency is shown on a logarithmic scale

Fig 9 Audibility curves for cat and man The audibility curve for cat is a best estimate based on all of the data shown in fig 8 The minimum audible field (MAF) shown for man is a calculated curve presented by Sivian and White (1933) for binaural listening with random incidence of the sound The curve for minimum audible pressure (MAP) shown for man is based on recent data given by Corso (1958)

The Audibility Curves

The results of all investigations of the cat s auditory thresholds that have been done in calibrated sound fields are shown in Fig 8 In each case the plotted points are the medians of the values given by the original authors Two sets of data are new The filled circles at the nine one octave steps from 125 to 32000 cps are the median thresholds of the present group of thirty four cats The filled squares at 350, 700, and 1400 cps are median thresholds based on tests of eight of the thirty four cats

Examination of Fig 8 shows that the results of the present study are in good agreement with the results from other laboratories This agreement cannot be ascribed to bias introduced by the testers, for the threshold determinations were made in terms of attenuation the associated SPL values were not known to the testers

Differences among the several experiments do not seem to be consistently related to the type of apparatus or to the type of reinforcement Similarly the type of animal preparation, binaural or monaural, does not appear to differentiate these functions Indeed, when the many possible sources of error are considered, the overall agreement among the several studies is quite good

After careful study of the several sources of data, the threshold values

index of sensitivity and that any additional connotation is questionable. The hit rate (response rate to tones) in a simple yes-no experiment is dependent on the false alarm rate (rate of spontaneous responses) as described by an operating characteristic. Now, even though the procedures of the present experiment are much more complicated than the simple yes-no experiment the cat's hit rate is undoubtedly dependent on his false alarm rate, and thus his threshold values will be dependent in some complex way on his false alarm rate. Moreover, it is likely that an animal's false alarm rate is more closely related to the immediate experimental context than is the false alarm rate of the human listener. For, while the animal's behavior is oriented to the avoidance of shock, the human listener's behavior is usually oriented by the verbal concept of "hearing" given in the instructions. Thus, it may be more important to control and describe the hit and false alarm rates in the case of sub-human listeners than it is in the case of human listeners.

When the hit and false alarm rates are specified at threshold values of the signal, in a sense, the cat's criterion is described. The procedures of the present experiment (see Method III, step 6 in Chapter II) represent a move in the direction of better control and description of these response rates, and in this spirit the following data were collected. The proportion of responses to tones presented at the approximate threshold was 0.185. The proportion of responses to possible artifacts was 0.038. And, the proportion of responses on noise-alone trials was 0.044. Each of these proportions was based on 1530 trials.

Under the assumption that these data could be treated as describing a point on an operating characteristic, the threshold levels given in this paper can be interpreted as levels which have a detectability index, d', of about 1.7. A clear explanation and definition of this index is given by Egan, Greenberg and Schulman (1961). The advantage of describing the threshold value in terms of a constant detectability index is that this number may be relatively independent of procedural variations.

Since the cats in our experiments could detect a tone generated by a 10-microvolt potential across the loudspeakers' voice coils, the possibility of artifacts of similar magnitude could not be ignored. Although artifacts were not audible to human listeners, it was still possible that the cat could hear them. The control apparatus for our audiometer, in fact, did produce clicks of very small magnitude in the audio circuits, and while these clicks could be observed on an oscilloscope, we were not able to measure them. In general, it should be remembered that artifacts are always associated with a tone presentation and that their elimination is a matter of degree. In relation to this problem, the closeness of the response rate to artifacts (0.038) to the rate on noise-alone trials (0.044) was an important finding, since it proved that the cats did not respond to artifacts when these artifacts were presented in isolation. Also, the audibility curves show that artifacts did not control the cat's response. Our experiments, however, do not eliminate the possibility that under some conditions artifacts could serve the function of a ready signal.

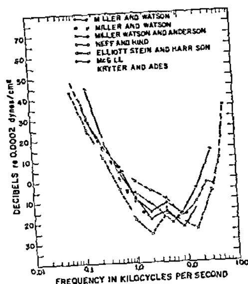


Fig 8

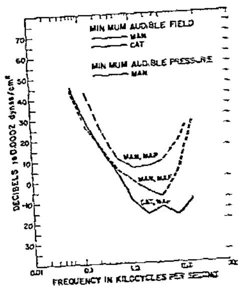


Fig 9

Fig 8 Audibility curves of cat Median results are shown for five previous sets of [illegible] calibrated sound fields and for two new sets of data (filled circles and squares). The [illegible] the median thresholds are shown in decibels re 0 0002 dynes cm² along the axis of ordinates [illegible] the abscissas are frequencies of the test tones shown in kilocycles per second. The [illegible] quency is shown on a logarithmic scale

Fig 9 Audibility curves for cat and man The audibility curve for cat is a "best estimate" [illegible] on all of the data shown in fig 8 The minimum audible field (MAF) shown for [illegible] ted curve presented by Sivian and White (1933) for binaural listening [illegible] the sound The curve for minimum audible pressure (MAP) shown for man is based on [illegible] data given by Corso (1958)

The Audibility Curves

The results of all investigations of the cat s auditory thresholds that [illegible] been done in calibrated sound fields are shown in Fig 8 In each case [illegible] plotted points are the medians of the values given by the original a[illegible] Two sets of data are new The filled circles at the nine one octave steps [illegible] 125 to 32000 cps are the median thresholds of the present group of [illegible] four cats The filled squares at 350, 700, and 1400 cps are median thresholds based on tests of eight of the thirty four cats

Examination of Fig 8 shows that the results of the present study [illegible] good agreement with the results from other laboratories This [illegible] cannot be ascribed to bias introduced by the testers for the thresh[illegible] minations were made in terms of attenuation, the associated SPL val[illegible] not known to the testers

Differences among the several experiments do not seem to be [illegible] related to the type of apparatus or to the type of reinforcement [illegible] the type of animal preparation, binaural or monaural does not ap[illegible] differentiate these functions Indeed, when the many possible sources [illegible] are considered, the overall agreement among the several studies is qu[illegible]

After careful study of the several sources of data, the thresh[illegible]

index of sensitivity and that any additional connotation is questionable. The hit rate (response rate to tones) in a simple yes-no experiment is dependent on the false alarm rate (rate of spontaneous responses) as described by an operating characteristic. Now, even though the procedures of the present experiment are much more complicated than the simple yes-no experiment, the cat's hit rate is undoubtedly dependent on his false alarm rate, and thus, his threshold values will be dependent in some complex way on his false alarm rate. Moreover, it is likely that an animal's false alarm rate is more closely related to the immediate experimental context than is the false alarm rate of the human listener. For, while the animal's behavior is oriented to the avoidance of shock, the human listener's behavior is usually oriented by the verbal concept of "hearing" given in the instructions. Thus, it may be more important to control and describe the hit and false alarm rates in the case of sub-human listeners than it is in the case of human listeners.

When the hit and false alarm rates are specified at threshold values of the signal, in a sense, the cat's criterion is described. The procedures of the present experiment (see Method III, step 6 in Chapter II) represent a move in the direction of better control and description of these response rates, and in this spirit the following data were collected. The proportion of responses to tones presented at the approximate threshold was 0.485. The proportion of responses to possible artifacts was 0.038. And, the proportion of responses on noise-alone trials was 0.044. Each of these proportions was based on 1530 trials.

Under the assumption that these data could be treated as describing a point on an operating characteristic, the threshold levels given in this paper can be interpreted as levels which have a detectability index, d_s, of about 1.7. A clear explanation and definition of this index is given by Egan, Greenberg, and Schulman (1961). The advantage of describing the threshold value in terms of a constant detectability index is that this number may be relatively independent of procedural variations.

Since the cats in our experiments could detect a tone generated by a 10 microvolt potential across the loudspeakers' voice coils, the possibility of artifacts of similar magnitude could not be ignored. Although artifacts were not audible to human listeners, it was still possible that the cat could hear them. The control apparatus for our audiometer, in fact, did produce clicks of very small magnitude in the audio circuits, and while these clicks could be observed on an oscilloscope, we were not able to measure them. In general, it should be remembered that artifacts are always associated with a tone presentation and that their elimination is a matter of degree. In relation to this problem, the closeness of the response rate to artifacts (0.038) to the rate on noise-alone trials (0.044) was an important finding since it proved that the cats did not respond to artifacts, when these artifacts were presented in isolation. Also, the audibility curves show that artifacts did not control the cat's response. Our experiments, however, do not eliminate the possibility that under some conditions artifacts could serve the function of a ready signal

IV EXPOSURE TO THE NOISE

Noise Field

The noise field for the deafness experiments was generated in a small reverberant room The components of the noise system were as follows a noise generator an attenuator a high pass filter whose cut off was at 250 cps and a 50 watt high fidelity amplifier Two driver units (University Loudspeakers Inc Type SA HF) mounted on each of two public address horns (University Loudspeakers Inc Type LH) served as the transducers Fig 10 shows the experimental arrangement for the exposure of the cats to noise After a few experiments hardware cloth cages were substituted for the restraining device pictured in Fig 10 because we feared that a cat would pull his head down into the collar and thus defend his ears from the sound

The intensity of the noise field was carefully calibrated in the region of the restraining device A General Radio Type 1551 A sound level meter was used for measurements of the overall level of the noise A spectral analysis of the noise was made with the same instruments as were used to calibrate the sound field of the audiometer With these instruments the noise was analyzed in ½ octave bands

In the region of the restraining device the maximum undistorted output of the noise system was 117 db and at ten surrounding positions the levels were within ±0 25 db of this value

The spectrum of the noise is shown by the solid line in Fig 11 The left hand ordinates are the levels for bands of 1 cps width expressed in decibels re the overall level and the right hand ordinates are the spectrum levels in SPL when the overall SPL is 115 db These measures were calculated from several analyses of the noise made with ½ octave bands Fig 12 shows the same data as the solid curve of Fig 11 plotted as octave band levels These figures show that the spectrum level has a maximum at about 750 cps and that it drops off slowly on either side of this value Notice that the octave band levels are about equal in the three bands centered at 850 1700 and 3400 cps

The broad band noise was used as the deafening stimulus in all experiments but one This exception is clearly noted in the text An estimate of the spectrum of the low frequency noise used in the exceptional experiment is shown by the dashed line of Fig 11 This dashed line is discussed in Chapter VIII

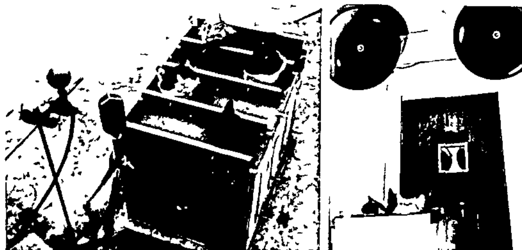


FIG 10 The experimental arrangement for the exposure of cats to the noise After a few experiments hardware cloth cages were substituted for the restraining device shown here, because it was feared that a cat would pull his head down into the collar and, thus, defend his ears from the sound The microphones and a recording sound level meter were used to monitor the sound levels during all exposures to the noise

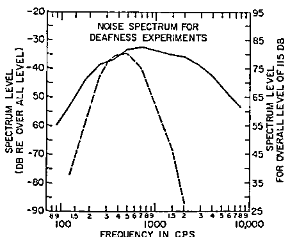


FIG 11

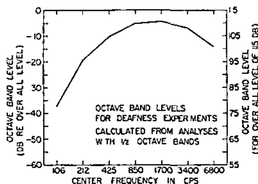


FIG 12

FIG 11 The spectrum of the broad band noise used in the deafness experiments is shown by the solid line The dashed line shows an estimate of the spectrum of a one octave band of low frequency noise used in the experiments of Chapter VIII The left hand ordinates are the levels in banks of 1 cps width expressed in decibels re the overall level, and the right hand ordinates are the spectrum levels when the overall SPL of the broad band noise is 115 db

FIG 12 Octave band levels of the broad band noise used in the deafness experiments

Procedure

All exposures to the broad band noise were administered as described here (A slightly different procedure was used for exposure of the cats to a low frequency band of noise, and this procedure is described in Chapter VIII) The cats were placed in the reverberant room, and they were restrained either in the device shown in Fig 10 or in individual hardware cloth cages In this manner one to six cats could be exposed simultaneously It should be noted that the cats were *not* anesthetized and that no other special procedures were used to prepare them for the exposure When the cats were settled in the room, the noise was turned on slowly over a period of 7 sec, at the completion of the exposure, it was turned off over the same period of time Throughout an exposure the noise level was monitored by a microphone and it was registered on a sound level recorder

Cat's Behavior

During an exposure the behavior of the cats was observed through a one way glass There were *no* dramatic responses to the noise At the onset of the noise there was a mild increase in activity Behavior such as circling in the cage or loud yowling was noticed After a few minutes they became quieted either sitting or lying down During the longer exposures of 2 hr or more, many of the cats appeared to go to sleep Of those that appeared to be asleep some were aroused when the experimenter turned off the noise and entered the room Others were not aroused until the experimenter gently shook their cages and a few were not aroused until they were touched It may be that these cases of apparent deep sleep were in part a result of the severe deafness present at the time Anthony Ackerman, and Lloyd (1959) have described the behavior of mice, rats, and guinea pigs during exposure to noise at about the 140 db level These authors found a decrease in activity during this intense exposure

V. TEMPORARY THRESHOLD SHIFTS

Experiments on temporary threshold shifts (TTSs) were conducted on cats for the following reasons (*a*) since in the case of man the general characteristics of noise induced TTS are well established, similar information for the cat could provide one basis for comparison of the deafening effects of noise on these two species, and (*b*) it was hoped that some of the relations between temporary and persistent threshold shifts could be clarified by a comparison of these two effects in the case of the cat

Temporary threshold shifts are available for three groups of cats that were exposed to similar conditions Group A, composed of seven cats, and Group B, composed of four cats, were exposed to the noise at an overall SPL of 105 db Group C, composed of five of the cats used in the preliminary experiments (see Chapter II), was exposed at 103 db For these three groups the duration of the exposure was ¼ hr Unlike all other cats used in these experiments, the cats of Group A were binaural

In another experiment (see Chapter VII) three groups of four cats were exposed to the noise at an overall SPL of 115 db for ⅛ hr One of these groups was tested extensively, and their results are reported below

In all of these TTS experiments, the order of test frequencies was counterbalanced for the post exposure tests, while prior to exposure it was randomized

TTS *Audiograms*

As representative of the effects of the ¼ hr exposure at 105 db, TTS audiograms at several times after exposure are shown for Group B in Fig 13 The TTS audiograms of Groups A and C, not shown, were in close agreement with those of Group B Fig 14 shows the TTS audiograms resulting from the ⅛ hr exposure to the 115 db level While the shapes of the audiograms shown in Fig 14 are typical, we believe that the absolute values of the shifts are atypically large Taking into account all of the data for this condition, we believe that a typical cat would have TTSs similar to those induced by the 105 db exposure In other words, it is our opinion that the ⅛ hr exposure at 115 db and the ¼ hr exposure at 105 db are nearly equivalent exposures for the typical cat

The findings shown in Figs 13 and 14 can be summarized as follows (1) Sizable TTSs are limited to the frequency region between 2 0 and 16 kc (2) The maximum TTS was measured at 4 kc (3) Because of its importance for the comparison of temporary and persistent threshold shifts, the relation between TTS at 2 0 and 8 0 kc should be noted TTS was greater at 8 0 kc

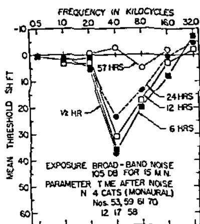


Fig 13

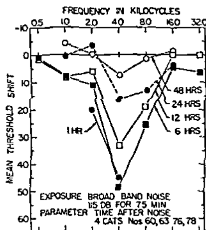


Fig 14

Fig 13 Temporary threshold shifts after exposure to the noise at an overall SPL of 105 db for ¼ hr The parameter is the time after noise in hours

Fig 14 Temporary threshold shifts after exposure to the noise at an overall SPL of 115 db for ¼ hr The parameter is the time after noise in hours

than at 2 0 kc for 14 of the 16 cats for which this comparison could be made (4) Since no TTS was measured at some frequencies, it can be inferred that the measured threshold shifts were not due to a general disruption of the cats' behavior in the threshold task (5) Recovery from TTS is orderly and complete, and it is discussed in more detail below

The above results confirm and extend the earlier findings of Lindquist, Neff, and Schuknecht (1954) Using a noise like that of the present experiments, these authors exposed cats at an overall level of 107 db for durations of 1/4, 1/2, or 1 hr As in the present experiments, large values of TTS were found and the maximum shift was at 4 kc

Recovery from TTS

Ward and Associates (1958, 1959*a*, 1959*b*) have shown for man that recovery from TTS at 4 kc follows a logarithmic course for a wide range of conditions Specifically, this logarithmic course is expected when (*a*) the time after exposure exceeds 2 min and (*b*) the TTS at 2 min after exposure (TTS_2) is less than about 40 db When TTS_2 is greater than about 40 db the recovery follows a quite different course Ward (1960) and Miller and Associates (1958) have described the recovery from these high values of TTS for man and cat, respectively and their results are shown in Fig 15 The dashed curve shows the course of recovery for two of the human subjects (Ward, 1960, Fig 1, Subjects HH and MB) To produce TTS_2 of 40 to 50 db, one of these human subjects was exposed for about 36 min, while the other required about 132 min for both listeners the noise was the octave band from 1200–2400 cps

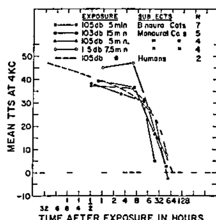


FIG 15 Recovery from high values of TTS at 4 kc The ordinates are TTSs in decibels while the abscissas are times after exposure in hours on a logarithmic scale Data for cat are shown by the symbols and similar data for man are represented by the dashed line See text for explanation

The symbols are means for the several groups of cats that were described earlier in this chapter It is apparent that the course of recovery from these high values of TTS is nearly identical for cat and man Note that, in terms of log time, the recovery is initially very slow and that it gradually increases in rate As Ward (1960) points out, the recovery from high values of TTS can be described as a linear function of time when the time after exposure exceeds one to two hours During this latter period the rate of decline of TTS is about 0 8 db/hr

Comparison of TTS *for Cat and Man*

When man is exposed to a broad band of thermal noise, it is generally true that TTS is greater for high than for low frequencies and that TTS often has a maximum at about 4 kc The data reported here show that similar features are found for the cat It has been demonstrated, in addition, that the course of recovery from high values of TTS is the same for both cat and man Therefore, it is probably true that the qualitative characteristics of TTS and its underlying processes are the same for these two species A quantitative comparison is given below

Although an experimental comparison of cat and man was not made in our laboratories, it was possible to make an estimate of the quantitative differences between cat and man For this comparison, only TTS at 4 kc was considered and the 15 min exposure at the overall SPL of 105 db was taken as a reference exposure The equations and constants given by Ward, Glorig, and Sklar (1959*a*) were used to calculate the expected results for man and the required changes in the reference exposure, if man were to have temporary threshold shifts equal to those for the cat The calculated values obtained for man can be regarded as close approximations to empirical values for exposures at SPLs of 106 db or less and for durations from several minutes to several hours since the equations are based on data provided by such exposures The exact values obtained by extrapolations to SPLs above 106 db are tentative

Table 4 *Comparison of TTS for cat and man*

	TTS_2	TTS_{60}	Required SPL if duration is 15 min and $TTS_2 = 44$ db	Required duration if SPL = 105 db and $TTS_2 = 44$ db
Man	22 5 (cal)	9 9 (cal)	123 db (cal)	180 min (cal)
Cat	44 0 (est)	38 0 (obs)	105 db	15 min
Diff in db	21 5	28 1	18 db	11 db

After the reference exposure, the temporary threshold shifts at 2 min after exposure (TTS_2) and at 60 min after exposure (TTS_{60}) were calculated for man These values were $TTS_2 = 22$ 5 db and $TTS_{60} = 9$ 9 db Sixty minutes after the reference exposure, the cat's temporary threshold shift (TTS_{60}) is 38 db The cat s TTS_2 was estimated by extrapolation to be 44 db Therefore, TTS_2 and TTS_{60} for the cat are greater than those for man by 21 5 and 28 1 db, respectively

If man s TTS_2 were to equal the cat's estimated TTS_2 of 44 db, it was calculated that the reference exposure would have to be increased for man by either 18 db in SPL or by 11 db in time The results of these calculations at the test frequency of 4 kc are summarized in Table 4

On the basis of the comparisons given above it is hypothesized that (*a*) the qualitative characteristics and the underlying processes of noise induced TTS are the same for cat and man, and (*b*) a given exposure will produce *more* TTS in cat than in man As a first approximation, it appears that in order to produce equivalent TTS in cat and man, the noise level for man must be 18 db higher than that for cat

VI PERSISTENT THRESHOLD SHIFTS AFTER CONTINUOUS EXPOSURE TO NOISE

In the experiments to be described below all cats were exposed to the same spectrum (Figs 11 and 12 of Chapter IV) the duration of the exposure was the variable The overall SPL of the broad band noise was 115 db while the duration of the exposure was ¼ ½ 2 or 8 hr

Procedure

Preliminary experiments Cats were exposed to the noise for durations of ¼ ½ and 2 hr Each cat was tested frequently and regularly throughout its post exposure life but no one schedule was used for all cats All of these cats were killed in order that their cochleas could be studied by histological methods The length of their post exposure lives varied from 85 days to 181 days The details of these preliminary experiments are given in another report by Miller and Associates (1958)

Later experiments Since the results of the preliminary experiments could be questioned on the grounds that the cats had been treated with dihydro streptomycin see Chapter II these experiments were repeated and extended using cats that had no treatment with an oto toxic drug In these later experiments 3 cats were exposed for ½ hr 8 cats[1] exposed for 2 hr and 6 cats were exposed for 8 hr As in the case of the preliminary experiments the overall SPL of the noise was 115 db

A standardized schedule of test sessions was used for all cats in these later experiments A test session consisted of measurements of a cat s threshold for the frequencies at each of the nine octave steps from 125 to 32 000 cps In each test session thresholds were determined at all nine frequencies and the order of test frequencies was randomized

In order to stabilize the data results from several test sessions were averaged Five test sessions were completed before a cat was exposed to the noise and the cat s pre exposure threshold was defined at each frequency as the mean of the five determinations The schedule of test sessions during a cat s post exposure life is shown in Table 5 Note that test sessions were conducted daily during the first two post exposure days and that thereafter tests were conducted at about ¼ ½ 1 2 and 3 lunar months after the exposure Note

[1] Four of these cats Nos 60 63 6 and 8 had previously been exposed to noise as described in Chapter VII Spaced exposure to noise These previous exposures had no persistent effects on their audiogram

TABLE 5 *Schedule of post-exposure tests and definitions of thresholds*

Defined threshold	N	Time after exposure of test sessions[a]
0 day	1	2–6 hr[b]
1 day	1	24 hr
2 day	1	48 hr
7.5 day	2	7 and 8 days
14 day	3	13, 14 and 15 days
28 day	3	27, 28, and 29 days
56 day	3	55, 56, and 57 days
84 day	5	82, 83, 84, 85, and 86 days

[a] The results in a given row were averaged for the calculation of thresholds and threshold shifts

[b] Not run in all cases

that the number of determinations per defined threshold varies with time after the exposure. A threshold shift at a particular frequency is defined as the difference between the pre exposure and the appropriate post exposure threshold described in Table 5.

All of the cats in these later experiments were perfused for histological preparation within 1 to 7 days after the last test session, thus, their post exposure lives were about 90 days.

Definition of PTS *and* $\overline{\text{PTS}}$

The threshold shifts at 84 days after exposure are referred to in the text as persistent threshold shifts (PTSs). It is claimed that recovery is completed by 84 days and that the thresholds obtained at 84 days represent the new persisting state of the cat's auditory sensitivity. This claim is strongly supported by the data on recovery and by the histological findings. In the case of a cat used in the preliminary experiments, each cat's final thresholds were used to define his PTSs, and the time at which these final thresholds were measured ranged from 75 to 171 days after the exposure.

It is convenient to use a single statistic in order to summarize the PTS audiogram. For the purpose of this monograph a statistic, $\overline{\text{PTS}}$, was defined as the mean PTS for the frequencies at the eight octave steps from 125 to 16,000 cps. Shifts at 32 kc were not included in this measure, since not all of the cats were tested at this frequency. It will be shown that $\overline{\text{PTS}}$ behaves in an orderly fashion and that for the conditions of these experiments the audiogram and the histological findings can be predicted when $\overline{\text{PTS}}$ is known.

Growth of Persistent Threshold Shifts

The relation between $\overline{\text{PTS}}$ and the duration of the exposure to noise is shown in Fig. 16. On this graph the abscissas are the durations of the ex-

Table 6 *Persistent threshold shifts after continuous exposure to noise*

Shown for each duration of the exposure are the mean PTSs. The standard errors of the mean based on both inter-cat variance, σ_m, and intra-cat variance, σ_R, are also shown.

Duration	Number of cats: Prelim	Later on	Total		Frequency in kc/sec: 0.125	0.25	0.5	1.0	2.0	4.0	8.0[a]	16.0	32.0[b]	$\overline{PTS}$[c]
				PTS	−2.1	1.2	4.0	19.2	13.8	10.8	2.3	3.5	—	5.6
¼	3	—	3	σ_m	2.0	3.1	4.8	5.4	9.8	9.0	3.9	3.1	—	3.2
				σ_R	1.9	1.9	1.9	1.9	2.5	2.5	2.5	2.5	—	0.8
				PTS	−0.2	1.9	4.9	13.5	25.3	20.4	6.3	−0.6	1.3	8.5
½	3	3	6	σ_m	2.5	2.5	2.3	3.5	6.9	9.5	4.8	3.6	5.2	2.6
				σ_R	1.4	1.4	1.4	1.4	1.8	1.8	1.8	1.8	1.8	0.6
				PTS	12.5	18.1	32.3	47.1	51.5	50.6	40.0	27.6	38.8	35.0
2	4	8	12	σ_m	2.5	3.3	5.6	4.3	2.6	3.1	5.3	7.1	11.9	3.2
				σ_R	1.0	1.0	1.0	1.0	1.2	1.2	1.2	1.2	1.2	0.4
				PTS	17.7	19.8	32.2	56.6	60.9	52.3	46.3	39.1	33.2	40.6
8	—	6	6	σ_m	3.7	1.2	7.4	8.9	4.8	6.2	12.8	12.0	13.6	6.7
				σ_R	1.4	1.4	1.4	1.4	1.8	1.8	1.8	1.8	1.8	0.6

[a] The cats in the preliminary experiments were tested at 8.8 kc rather than 8.0 kc

[b] The test frequency of 32.0 kc was not used in the preliminary experiments

[c] Mean is the mean of the frequencies from 0.125 to 16.0 kc

Audiograms

The PTS data resulting from these continuous exposures are shown in the conventional form of audiograms in Fig. 19. Inspection of this figure shows that the largest shifts are found between 1.0 and 4.0 kc. It should be noted that unlike the case of TTS, the loss at 2.0 kc is consistently greater than the loss at 8.0 kc. This display of the data clearly shows again that the major effect of increasing the duration of the exposure was between the ½ hr and the 2 hr durations. The properties of the audiogram will be discussed in more detail in Chapter X.

Table 6 summarizes the PTS data that have been presented above (Figs. 16, 17, 18 and 19). This table shows the composition of the groups, the mean PTS for each frequency, as well as $\overline{PTS}$. Below each mean two measures of variability are given. The first, labeled σ_m, is the standard error of the mean based on the estimated inter-cat variance. The second, labeled σ_R, is an estimation of the reliability of the mean for the particular cats of these samples; in other words, it is an estimate of the standard error of the PTS based on the intra-cat variability. Examination of these measures of variability shows that while these means are precisely determined for the particular cats of these experiments, the enormous inter-cat variability precludes precise estimates of the population means from these small numbers of cases.

TABLE 7 *Mean injury ratings for cochleas of cats exposed to the broad band noise*

Duration of noise (Hr)	Number of cats: pre lim	Number of cats: later		(Mean)	Location of injury: III	II			I		
					X	U	M	L	U	M	L
				mm from R W	20.5	17.0	15.3	13.5	10.8	7.5	4.5
				Equivalent frequency in kc/sec	0.15	0.7	1.09	1.7	3.4	7.6	17.0
¼	2	—	I	1.87	1.50	2.00	2.75	2.25	2.00	2.00	1.5
			σ_m	0.18	0.0	0.50	0.25	0.25	0.50	0.0	0.0
½	3	3	I	2.04	1.50	1.83	2.17	2.42	2.06	2.33	1.75
			σ_m	0.16	0.13	0.11	0.17	0.21	0.34	0.40	0.28
2	4	8	I	2.88	1.79	2.13	2.88	3.67	3.83	3.04	2.88
			σ_m	0.16	0.11	0.14	0.19	0.24	0.28	0.28	0.33
2[a]	-10-		I	2.41	1.70	2.00	2.50	3.25	2.85	2.80	1.75
			σ_m	0.31	0.11	0.13	0.17	0.08	0.24	0.38	0.15
8	—	6	I	3.36	2.17	2.75	3.33	4.08	4.00	3.67	3.5
			σ_m	0.21	0.20	0.17	0.21	0.20	0.26	0.36	0.71

[a] Binaural and untrained with short post exposure life

As previously mentioned, the inter cat variability is due largely to the noise exposure, since prior to exposure the inter cat variance in thresholds is smaller than after exposure by a factor of about 16 The intra cat variance on the other hand is relatively unaffected by the noise For an individual cat, the largest increase in intra cat variability was a factor of 10, the increase of the mean intra cat variance from before exposure to 84 days after it was 1 26 db

Ratings of Cochlear Injuries

The methods for the preparation of the cochleas for histological examination and the rating scale used as a measure of the degree of injury are given in Chapter IX The analysis of the relations between the behavioral findings and the histological findings is given for all exposures to the broad band noise in Chapter X Nevertheless, the mean injury ratings will be presented here for the continuous exposures to the broad band noise

Table 7 shows the means of the injury ratings, which can vary from 1 0 for a normal end organ to 5 0 for complete destruction of the external hair cells at each of several positions along the basilar membrane It should be understood that these positions are only approximate and that the rating was based on an examination of the indicated region not a particular distance from the round window In Table 7 the positions along the basilar membrane are indicated Below the name of each location the approximate distance

from the round window is given in millimeters and the corresponding test tone frequency as determined by Schuknecht (1953) is also shown

Examination of Table 7 reveals that the greatest injury is found in the lower and middle portions of Turn II and the upper and middle portions of Turn I It can be seen that as the duration of the exposure increases the severity of the injury increases Also the longer the exposure the greater the spread of the injury along the basilar membrane

Note that a group of binaural untrained cats is included in Table 7 This group was used to control against the possibility that some aspect of our laboratory procedures increased the cats susceptibility to noise For example it was feared that perhaps the destruction of one ear or the laboratory diet or the training procedure in some unknown way made the cats susceptible to the noise exposure In order to control against these possibilities seven cats were exposed to the noise on the very same morning that each was received from the dealer These cats were perfused between 2 and 4 hours after the cessation of the noise Two of the cats were discarded because evidence of middle ear infection was observed during post mortem examination Ten ears from five cats were examined and rated for cochlear injury The results in Table 7 show that injury was present in these cochleas although the ratings are not quite so high as those for the trained cats with long post exposure lives

Overall the histological findings are consistent with the behavioral findings Permanent injuries inferred from the behavioral findings were in fact observed Untrained cats that had never lived in the laboratory showed injury patterns that were similar to those observed for trained cats Differences that do exist in the histological findings for trained and untrained cats can probably be attributed to differences in the lengths of their post exposure lives See Chapters IX and X for more detailed accounts of the histological findings and their relations to the audiograms

Recovery

Temporary threshold shifts (TTSs) that are induced by exposure to sound are defined as those threshold shifts which recover to zero Persistent or permanent threshold shifts (PTSs) that are induced by sound are those stable threshold shifts that remain after the completion of recovery

In a case where noise exposure produces PTS we believe that threshold shifts measured before recovery is complete should be distinguished from PTS This distinction is proposed on the general grounds that recovery from an exposure to noise which has produced a new and permanent injury may involve complex interactions among fatigue processes temporary injuries and permanent injuries which differ from those factors controlling recovery from pure TTS This distinction also is proposed on the specific grounds that for the conditions that we have studied pure TTS has different frequency [illegible] pure PTS For these reasons we shall use the

term compound threshold shift (CTS) to describe threshold shifts that have both temporary and new permanent components

While recovery from TTS has been extensively studied by Ward Glorig and Sklar (1958 1959*a* 1959*b*) and others only few data on the recovery from the temporary components of CTSs have been available Knowledge of the course and duration of the recovery from CTS to the final PTS level is required for the design of experiments the practical decisions of hearing conservation and for theories of the recovery process Moreover it is obvious that before a threshold shift can be asserted to be permanent the recovery from the temporary components of the CTS must be shown to be complete

Initial data on the recovery from the temporary components of the CTS were obtained during the preliminary experiments After a single exposure to noise the cats in these experiments were tested periodically throughout their post exposure lives which ranged from three to six months Recovery followed a regular and decelerating course the major portion of the recovery occurred within one to five weeks after the exposure to noise and generally recovery was completed in the first two post exposure months While in most cases the post exposure thresholds were remarkably stable this was not true in all cases At some frequencies some of the cats did recover an additional 8 to 13 db during the third and fourth post exposure months Because of several difficulties encountered in these preliminary experiments we doubt that these cases of additional recovery were due to auditory processes

Our most reliable and extensive set of data on the recovery from the temporary components of CTS were obtained during the later experiments Recovery curves for the test frequencies of 0 25 2 0 and 8 0 kc are shown in Fig 20 as examples of these results On each panel of this figure the abscissas are the times after exposure in days while the ordinates are the mean threshold shifts in decibels The parameter is the duration of the exposure to the noise A hat over a symbol means that at least one cat in the group had a threshold shift so large that he did not respond at the audiometer s maximum output The dotted lines which connect the symbols with hats represent lower bounds on these mean recovery curves The broken lines and open symbols represent data from the preliminary experiments while the solid lines and solid symbols represent data from the later experiments

The recovery curves for other test frequencies are not shown since they were highly similar to those shown in Fig 20 For all frequencies recovery is extremely rapid during the first few post exposure days and thereafter the rate gradually slows Also the duration of the period during which recovery can be observed seems to increase with the duration of the exposure In almost all cases the curves have become horizontal by 84 days after the exposure

Since during the later experiments test sessions were run at regularly scheduled times after the exposure the recovery from threshold shifts will be examined in more detail for these experiments

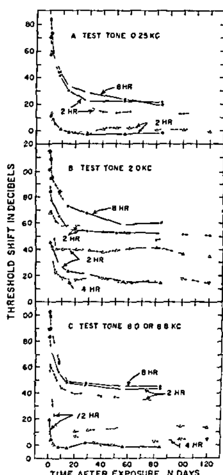


FIG. 20 Recovery of the threshold shift after exposure to the broad band noise at an overall SPL of 115 db. The parameter is the duration of the exposure to noise. The abscissas are the times after exposure in days while the ordinates on each panel are the mean threshold shifts in decibels. Dashed curves and open circles are based on data obtained during the preliminary experiments. The solid points are mean values of threshold shifts measured at the indicated times after exposure during the later experiments. A bar over a symbol means that at least one cat in the group had a threshold shift so large that he could not respond at the audiometer's maximum output. In such cases the maximum output was used to calculate the lower bound on the threshold shift.

The data of these later experiments were examined to determine whether or not recovery had stopped by 56 days after the exposure. For each cat and test tone frequency the difference between the 56 and 84 day threshold shifts was examined, using the standard *t* test, for statistical significance at the 5% level of confidence. Since of 288 tests only 13 proved significant, it seems unlikely that recovery was continuing beyond 56 days after the exposure. The change in the threshold shift during the period from 56 to 84 days after exposure was calculated for all frequencies and cats. Mean values of this [illegible] db for the 4 hr exposure, 0.46 db for the 2 hr exposure [illegible] db for the 8 hr exposure. Thus on the basis of all the

TABLE 8 *Amount of recovery and the duration of the recovery period*

For each exposure condition the mean difference between the CTS measured 1 day after exposure and the PTS measured 84 days after exposure is shown Also shown are the mean number of days to recover with 6 db of $\overline{PTS}$ Only the data of the later experiments are used

Exposure duration		Frequency in kc/sec									Mean (125–16 000 cps)
		0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	
¼ hr	1d–84d	8.0	17.1	22.2	33.7	30.9	33.3	20.4	17.2	24.8	23.1
	Days of recovery (N = 3 cats)	12.3	7.5	9.9	19.2	26.1	10.8	4.0	1.8	2.0	11.1
2 hr	1d–84d (lower bound)	41.1	47.8	50.8	43.1	32.4	40.8	34.9	30.3	35.6	39.2
	Days of recovery (N = 4 cats)	23.3	20.1	27.7	17.5	18.9	16.6	17.9	18.5	22.7	18.7
8 hr	1d–84d (lower bound)	48.6	52.5	61.1	51.6	47.2	48.1	52.5	36.2	47.6	49.7
	Days of recovery (N = 6 cats)	31.3	40.4	34.8	40.1	28.0	31.8	21.8	34.1	36.8	32.9

recovery data that we have obtained we believe that recovery from CTS is complete by two to three months after exposure and that for these exposure conditions cases of recovery during the fourth post exposure month are rare, if not artifactual

The relation between the time to recover and the duration of the exposure was also examined Since it is difficult to determine the time at which a recovery curve has reached its asymptote, the following statistic is defined as the period of recovery This statistic is the number of days required to recover within 6 db of the threshold shift at 84 days that is, the number of days for threshold shift to fall within 6 db of the PTS For the cats used in the later experiments this measure was obtained for each of the test tone frequencies

A related variable is a measure of the amount of recovery It is defined as the difference in decibels between the threshold shift at 1 and 84 days after the exposure This difference of course, is the magnitude of the temporary component of the compound threshold shift at the indicated time In most cases it was impossible to measure thresholds one day after exposure, since the cat would not respond at the highest intensity of the audiometer Nonetheless a lower bound could be obtained on the amount of recovery

Table 8 shows both the period of recovery and the amount of recovery as they were defined above The duration of the recovery period increases with the duration of the exposure The periods of recovery for the average thresh

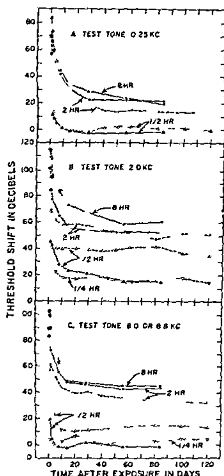


FIG 20 Recovery of the threshold shift after exposure to the broad band noise at an overall SPL of 115 db The parameter is the duration of the exposure to noise The abscissas are the times after exposure in days, while the ordinates on each panel are the mean threshold shifts in decibels Dashed curves and open circles are based on data obtained during the preliminary experiments The solid points are mean values of threshold shifts measured at the indicated times after exposure during the later experiments A hat over a symbol means that at least one cat in the group had a threshold shift so large that he could not respond at the audiometer's maximum output In such cases the maximum output was used to calculate the lower bound on the threshold shift

The data of these later experiments were examined to determine whether or not recovery had stopped by 56 days after the exposure For each cat and test tone frequency the difference between the 56 and 84 day threshold shifts was examined using the standard *t* test, for statistical significance at the 5% level of confidence Since of 288 tests only 13 proved significant, it seems unlikely that recovery was continuing beyond 56 days after the exposure The change in the threshold shift during the period from 56 to 84 days after exposure was calculated for all frequencies and cats Mean values of this [illegible] were 0 54 db for the ½ hr exposure 0 46 db for the 2 hr exposure [illegible] db for the 8 hr exposure Thus on the basis of all the

tions of our experiments which produced PTSs for cat would not produce PTS for man In particular Davis and Associates (1950) report data for men exposed to noise similar to the one we used to deafen cats Overall SPLs of 110 120 and 130 db and durations from 1 to 32 min were used also one exposure at an overall SPL of 120 db had a duration of 64 min While large TTSs were found no persistent threshold shifts resulted from these exposures

It will be recalled that on the basis of a comparison of TTS for cat and man it was estimated that man would require for the same TTS as cat an exposure about 18 db greater than that for the cat If this difference be correct for susceptibility to permanent injury, then 133 db would produce results for man similar to those for cat This prediction does not seem at variance with the known data for man

The difference between cat and man in susceptibility to PTS can be estimated from known properties of their ears and hearing Above 500 cps the cat s auditory sensitivity to tonal sound fields is at least 8 db better than the comparable sensitivity of man If this difference is due to properties of the external and middle ears of these animals then in a given sound field at least 8 db more energy is reaching the inner ear of cat than that of man But once in the cochlea a greater bandwidth is mapped onto a millimeter of the cat s basilar membrane than onto a millimeter of man s basilar membrane as inferred by Elliot Stein and Harrison (1960) Greenwood (1961) and Watson (1963) In the case of a broad band noise this factor could increase the exposure of cat over that for man by a factor equivalent to an SPL change of about 3 0 to 7 0 db Thus 11 or more db of the tentative 18 db difference between cat and man can be accounted for by the acoustic properties of the external middle and inner ears

Hypothetical Growth of PTS and Related Variables

Several important variables have been related to the duration of the exposure to noise These are the amount of PTS the size of the temporary component of CTS at any particular time after exposure and the time required for recovery from the temporary component of CTS Unfortunately the relations between these variables and the exposure duration are complex and not completely determined by our experiments Our best estimates of these relations are given in the text below and illustrated in Fig 21 The discussion below is limited to exposures to the broad band noise at the 115 db level and only test tones in the mid frequency range from 1 to 4 kc are considered

We believe that post exposure threshold shifts cannot exceed those measured in the presence of the noise that is the masking produced by the noise It is possible to estimate the amount of masking produced by the broad band noise using measurements made by Watson (1959) The horizontal line at about 117 db in Fig 21 indicates the amount of masking for the conditions under consideration and it reflects our assumption that the amount of masking

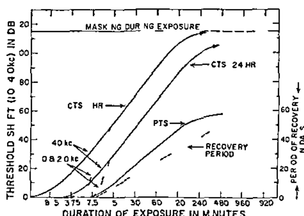


FIG 21 Hypothetical growth of CTS PTS and the period of recovery for a typical cat exposed to the broad band noise at an overall SPL of 115 db Although the lines of this graph are hypothetical they are drawn in a fashion consistent with the available data The abscissas are the durations of the exposure in minutes placed along a logarithmic scale The left hand ordinates are the threshold shifts in decibels while the right hand ordinates are the periods of recovery in days

places an upper bound on post exposure threshold shifts Compound threshold shifts measured one or twenty four hours after cessation of the noise are shown approaching the assumed upper limit Note nonetheless that throughout a broad range of exposure conditions both CTS and TTS are shown as linear functions of the logarithm of exposure duration

If the duration of the exposure is less than about 7 5 min only temporary threshold shifts are observed As the duration of the exposure increases to 15 min permanent threshold shifts are induced and the shifts become on the average equally severe for each of the mid frequencies The initial branching of the functions labelled CTS indicates that greater shifts are found at 4 kc than at 1 or 2 kc for those short durations of exposure that only produce TTS

It was noted earlier that the exact course of the growth of PTS cannot be determined from the present data Our best guess about this growth is shown in Fig 21 by the line labelled PTS Over its linear portion the slope of this curve appears to be less than that for CTS It is not known whether or not the PTS curve reaches a maximum One cat (No 90) did have PTSs as large as 78 db after an exposure of 8 hr perhaps more typical cats would reach PTSs of that size if the duration of the exposure were made sufficiently long

The number of days required for recovery from the temporary component of CTS is also shown in Fig 21 The reader should recall that the duration of the recovery period is defined here as the number of days required for the CTS to reach a level within 6 db of the PTS So defined the duration of the recovery period appears to grow as a linear function of the logarithm of the dur t f the exposure We conjecture that this function must reach and perha[ ] s thr ugh a maximum This conjecture follows from the

hypothesis that for any exposure spectrum only a given set of structural elements in the sensory system can be temporarily or permanently injured For sufficiently long durations of exposure, it is hypothesized that all elements which can be permanently injured have been so injured furthermore, it is suspected that only a few elements would remain that could be temporarily injured by the spectrum Thus for extremely long exposures, the temporary component of CTS would probably be small and recovery from it would be relatively quick

VII. PERSISTENT THRESHOLD SHIFTS FROM SPACED EXPOSURES TO NOISE

In this set of experiments the effect of breaking up a long continuous exposure into a series of short, spaced exposures was examined. Because it was known that exposure to the broad band noise for ½ hr at an overall SPL of 115 db produces little or no permanent injury, this duration was chosen for all of the short exposures in the series. Furthermore, because a continuous exposure to the noise at 115 db for 2 hr was known to produce sizable PTS, all cats were given 16 of the short exposures to noise and, thus, were exposed a total of 2 hours.

Four groups of four cats each were exposed according to the following schedules. Group I received a two hour exposure to the noise which was continuous in time. Groups II, III, and IV were exposed to the noise in sixteen, ½ hr doses. These doses were spaced, from onset to onset, at one hour intervals for Group II, at six hour intervals for Group III, and at 24 hour intervals for Group IV. Since the level of noise was constant for all groups and since all cats were exposed for a total of 2 hours, all cats were exposed to the same total energy.

Temporary and Compound Threshold Shifts During a Series of Exposures

The cats in Group IV *a*[1] were first used to determine the TTS audiogram for the single, ½ hr exposure, and these data were presented in Fig. 14 of Chapter V. About one month after the first exposure to the noise, a series of 16 of these ½ hr exposures which were spaced at 24 hr intervals, was begun on the cats of this group. These animals were tested at 2.0 and 4.0 kc before and after each exposure. These tests were accomplished during the one hour period preceding the exposure and during the one hour period that began 30 minutes after the exposure. For three control days prior to the beginning of the experiment the threshold tests were conducted, but there were no exposures to the noise.

The effects of this series of exposures are shown in Fig. 22. The threshold *shifts at 4.0 and 2.0 kc are shown in the upper and lower panels, respectively*

[1] The cats in Group IV had the following history of exposure to the broad band noise at 115 db: (1) one ½-hr exposure (Chapter V on TTS); (2) a series of 16 spaced exposures with an inter-exposure interval of 24 hr; (3) a series of 8 spaced exposures also at an inter-exposure interval of 24 hr [illegible]; (4) a continuous 2 hr exposure. Group IV is termed Group IV *a* for the exposures listed under (1), (2) and (3). These same four cats are called Group IV *b* for the exposure listed u[illegible]

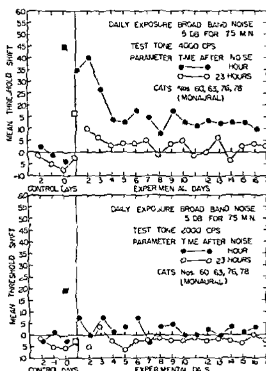
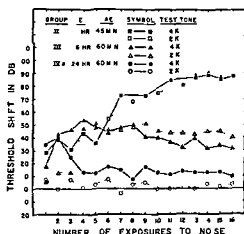


Fig 22 Threshold shifts measured before and after each of a series of exposures to the noise For this case the inter exposure interval (IEI) was 24 hr each exposure to the broad band noise was for 7.5 min at an overall SPL of 115 db The ordinates are the mean threshold shifts in decibels re the threshold before the beginning of the experiment The filled symbols in all cases are measures taken during the period from 30 to 90 min after exposure The open symbols represent threshold shifts measured approximately 23 hrs after an exposure The circles on the left hand portions of the graphs show the results of tests on three control days when the noise was not turned on The squares on the left hand portion are the comparable data points for these four cats after they had been given a single exposure to the noise for 7.5 min approximately one month prior to the series of sixteen successive exposures The results of this single previous exposure are shown in more detail in *Fig 14 in Chapter V*

Throughout this series of exposures the reference thresholds are the normal thresholds which were established prior to beginning the series The squares on the left hand portion of the figure show the results of the original ⅛ hr exposure to the noise which was given one month prior to the series of 16 exposures The circles on the left hand portion of the panels show the control results obtained when the noise is omitted To the right of the vertical line on each panel the results obtained *during the sequence of the 16 successive* exposures are shown The striking finding shown in Fig 22 is that the size *of the threshold shift* decreases during the series of exposures to noise Although this decrease is reliable it is not understood It may be that somehow the cats were becoming more resistant to the noise Or it may be that the cats suffer a post exposure tinnitus and that the decreases in the threshold



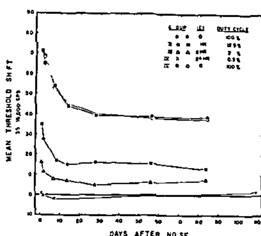


Fig 23 Fig 24

Fig 23 The growth of threshold shift during a series of sixteen exposures to the broad band noise Each exposure is at an overall SPL of 115 db for a duration of 7 5 min The parameters are the inter exposure intervals (IEI) and the frequency of the test tone

Fig 24 Recovery of the threshold after a series of sixteen exposures to the broad band noise Each exposure was at an overall SPL of 115 db for a duration of 7 5 min The abscissas are the days after the last exposure in the series while the ordinates are the threshold shifts averaged over the eight octave steps from 125 to 16 000 cps ($\overline{CTS}$) The parameter is the interval between the successive exposures Note *that for the continuous condition the symbols with hats should be taken as lower bounds on the sample means*

shift reflect the possibility that the cats learn to distinguish the test tone from the tinnitus

After a four month rest from the noise these cats were given a second series of eight exposures The schedule was the same as before and the results although they are not shown matched those of the last seven days of the first series If the decline of TTS during the first series represents a learning phenomenon then there was no forgetting after a four month rest After an additional one month rest these cats were exposed to the uninterrupted noise for 2 hours For the purposes of presenting the result of the continuous exposure this group is referred to as Group IV *b* (Because Group IV *b* and Group I received the same continuous exposure for 2 hr there were eight cats under this condition The data for these eight cats are presented here and in the previous chapter) It should be noted here that these cats in Group IV *b* were neither more nor less susceptible to the continuous exposure of 2 hr than those of Group I who had no previous exposure to the noise Thus if the cats in Group IV did learn to protect themselves from the short ⅛ hr exposures their learning did not help them in the case of the longer 2 hr exposure

The growth of threshold shift during the series of exposures is shown for all of the groups of this experiment in Fig 23 (The results for Group IV *a* previously shown in Fig 22 are also shown in Fig 23) For all groups these

measurements were made about one hour after each cessation of the noise. The solid symbols are for the threshold shifts at 4 kc, while the open symbols show the shifts at 2 kc. The squares, triangles, and circles represent inter-exposure intervals (IEIs) of one, six, and twenty four hours, respectively. First examine the results for the inter-exposure interval of 1 hr. During the initial four or five exposures, there is only a slight increase in the threshold shift. After exposure number five the threshold shift begins to increase. Note that as this series was continued the mean value of the threshold shift reached a lower bound of about 88 db. Because of the short time between exposures these cats were tested at 2 kc only after exposures No. 6, 7, 10, and 12. Note, however, that by exposure No. 6 the shift at 2 kc is large and about the same as the shift at 4 kc. Next examine the results for the inter-exposure interval of 6 hours. Note that the shift at 4 kc initially increases and then slowly decreases. In contrast with other groups, this condition results in roughly constant threshold shift at 4 kc. The shift at 2 kc, however, follows a different course. After exposures No. 1, 2, and 3 little shift is observed at 2 kc, but after the fourth exposure, the shift at 2 kc suddenly becomes about as large as the shift at 4 kc. While the significance of this jump in the threshold shift at 2 kc is unknown, it may mark the beginning of permanent injury.

Comparisons among the conditions for this portion of the experiment show that the threshold shifts measured during such a series of exposures may increase, remain approximately constant, or decrease.

Recovery and PTS *for Spaced Exposures*

The build up of the threshold shift during the series of exposures was shown above. Fig. 24 shows the recovery from threshold shift after the final exposure in the series. The top two curves display for the continuous exposures the recovery of the mean thresholds, as shown by the reduction in the mean threshold shifts. The bottom three curves show the recovery after the series with inter-exposure intervals of 1, 6, or 24 hr. It can be seen that orderly recovery curves, which are asymptotic to values above zero, are obtained for the topmost four curves, while the 24 hr spacing resulted in little or no $\overline{PTS}$. All of the data on recovery from the spaced exposures are consistent with those found for continuous exposures, that is, the more severe the threshold shifts, the longer the period of recovery. Recovery to an asymptotic value is complete in all cases by 56 days after exposure.

The relation between mean $\overline{PTS}$ and the inter-exposure interval is shown in Fig. 25. Symbols on this figure represent the results for individual cats and the mean of each group is shown by the line. Inspection of this curve shows that while an inter-exposure interval of one hour reduces the mean $\overline{PTS}$, the inter-exposure interval must be extended beyond six hours if all cats are to be protected from persistent threshold shifts. It is apparent then, that for exposure schedules similar to those used here, a very small duty cycle of the order of 1.0% or 0.5% is required in order to reduce $\overline{PTS}$ to

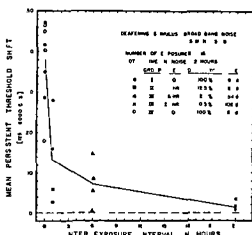


Fig 25

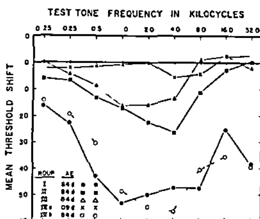


Fig 26

Fig 25 Reduction in $\overline{PTS}$ with increasing inter exposure interval The symbols show the results for individual cats while the lines connect the group means The abscissas are the inter exposure intervals in hours the ordinates are values of $\overline{PTS}$ in decibels The times after exposure at which the PTSs were measured are shown on the figure

Fig 26 Mean PTS audiograms for groups exposed to the broad band noise in 16 doses of 7 5 min in duration Inter exposure intervals of 0 1 6 and 24 hrs were used for Groups I II III and IV *a* respectively Group IV *b* was exposed continuously that is the inter exposure interval was zero

nearly zero for all cats An electrophysiological investigation by Eldredge, Covell, and Gannon (1959) supports this view For equated total exposure energy, a 25 % duty cycle and a 100 % duty cycle were equally injurious for intense exposures

It is interesting to note the equivalent continuous exposure for each of the inter exposure intervals The inter exposure intervals and their equivalent continuous durations are respectively as follows 0 hr 120 min 1 hr 48 min 6 hr, 24 min, 24 hr, 9 min These values were found by interpolation on Fig 16

The wide individual differences in susceptibility to the noise exposure should be noted As in the case of the continuous exposures these cats differed only slightly in their auditory sensitivity before exposure to the noise Although an analysis of variance allows us to infer that the downward trend which is present in the graph is present in the population, it is obvious that the course of this downward trend is only crudely established by these variable sample data

Table 9 shows the mean values of PTS for each of the conditions of the present experiment Also shown are the standard errors of the mean, σ_R and σ_m that are based on the intra and inter cat variances, respectively

Table 10 shows the injury ratings for these animals These findings are consistent with the audiometric data of this section

The mean audiograms for the several inter exposure intervals are shown

TABLE 9 *Persistent threshold shifts after spaced exposures to the broad band noise for each inter exposure interval (IEI)*

The standard errors of the means based on the inter-cat variance, σ_m, and the intra cat variance, σ_R, are also shown

Group	IEI		Frequency in kc/sec 0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	$\overline{PTS}$
		PTS	16.2	23.0	42.8	53.2	49.7	47.6	47.4	25.7	>38.2	38.2
I	0	σ_m	6.31	6.16	6.45	6.06	7.00	8.53	7.94	16.11	—	3.97
		PTS	14.6	19.6	30.2	48.7	54.9	56.7	40.4	35.9	>39.4	37.6
IV *b*	0	σ_m	2.69	5.37	11.44	12.60	4.31	2.57	9.83	11.60	—	6.63
		PTS	6.0	6.8	13.2	17.0	22.5	26.8	11.0	2.8	−0.8	13.2
II	1	σ_m	4.02	5.84	9.93	9.39	8.50	11.31	6.57	3.77	3.20	5.74
		PTS	0.2	4.8	9.2	16.8	16.0	14.0	−1.0	−2.25	2.25	7.2
III	*6*	σ_m	*1.44*	*1.59*	*4.27*	*9.99*	*9.70*	*7.94*	*0.82*	*2.18*	*1.38*	*3.06*
		PTS	2.2	2.0	1.1	0.8	0.0	5.9	4.2	−2.0	−2.4	2.3
IV *a*	24	σ_m	2.83	0.96	2.75	0.60	1.74	2.59	2.71	0.65	1.01	0.61

σ_R for $f \leq 1.0$ kc 1.7 db σ_R for $\overline{PTS} = 0.7$ db
for $f > 1.0$ kc – 2.2 db

in Fig 26 The shapes of these audiograms do not seem to differ significantly from those resulting from continuous exposure to noise It should be noted again that the major loss falls between 1.0 and 4.0 kc

One other interesting relation was suggested in these data Differences in PTSs among cats were positively correlated with their CTSs measured at 2 hr after the final exposure in the series This relation was noted for both the 1-hr and the 6 hr inter exposure intervals, but the intercept of the regression line depends on the experimental condition For example, if the average PTS for 2.0 and 4.0 kc be 30 db, then the corresponding CTSs measured at 2 hr after exposure would be about 55 db and 85 db for the 6 hr and the 1 hr inter exposure intervals, respectively Table 11 summarizes the data relevant to this point If these trends be true, then to predict PTSs from CTSs the exposure conditions would have to be specified

Discussion

The experiment reported above was designed to provide information concerning the effects of interspersing silent intervals between exposures to noise In the case of TTS it has been shown that rest intervals reduce the TTS present at the completion of a series, see for example Ward, Glorig, and Sklar (1958 1959c) But this finding is expected for TTS, since TTS by its nature and definition is a quantity which does, in fact, recover in the absence of sufficient stimulation In the case of PTS, it was not at all obvious that

TABLE 10 *Mean injury ratings for cochleas of cats exposed to the broad band noise for 2 hrs*

Data are shown for continuous and for spaced exposures with IEIs of 1 and 6 hr

				Location of injury						
				III	II			I		
				A	U	M	L	U	M	L
				mm from R W						
				20.5	17.0	15.3	13.5	10.8	7.5	4.5
	Number			Equivalent frequency in kc/sec						
IEI[a]	of cats		(Mean)	0.15	0.7	1.09	1.7	3.4	7.6	17.0
0[b]	12	*I*	2.88	1.79	2.13	2.88	3.67	3.83	3.04	2.88
		σ_m	0.16	0.11	0.14	0.19	0.24	0.28	0.28	0.33
1	4	*I*	2.25	1.63	2.13	2.75	3.25	2.63	2.0	1.38
		σ_m	0.29	0.12	0.24	0.48	0.32	0.37	0.31	0.24
6	4	*I*	1.77	1.50	1.88	2.00	2.13	2.00	1.63	1.25
		σ_m	0.14	0.20	0.12	0.35	0.24	0.20	0.12	0.14

[a] Histological data not available for animals with IEI of 24 hrs since these cats were given an additional continuous exposure

[b] Same data as those shown in table 7 for trained cats given a 2 hr exposure which was continuous

breaking up an exposure by rest intervals would reduce the total permanent injury For PTS by its definition is persistent and it is not clear that rest intervals should somehow allow recovery from it

Nevertheless, the data that have just been presented show that both CTS and PTS decrease with increases in the duration of the silent interval between successive exposures to the noise While trying to analyze this experiment it became apparent that an understanding or conception of the processes underlying the build up of PTS or injury during a series of spaced exposures depends on an understanding or conception of the processes underlying the results of the simpler experiment of increasing the duration of a continuous exposure to noise

Stochastic models Why does increasing the duration of an exposure to noise increase the amount of injury? While the answer to this question is not known it is clear that the role of the duration of an exposure depends upon the injurious action of the noise Two views of this action are sketched below and their implications are discussed One view is that during any very short period of time the probability of permanent injury to a particular sensory element of the inner ear depends on the spectral and intensive properties of the noise and that this probability is independent of the duration of the noise According to this view an increase in the duration of an exposure increases the opportunities for injury and therefore, the total number of

TABLE 11 *Compound threshold shifts and persistent threshold shifts after a series of spaced exposures to noise*

CTS is the mean of the CTSs measured 2 hr after exposure at 2 kc and 4 kc The PTS is the corresponding average of the threshold shifts 84 days after exposure, while $\overline{\text{PTS}}$ is the average across frequencies Results are shown for *inter-exposure intervals of 1 and 6 hr*

I E I	CAT	CTS_2 hr	PTS	$\overline{\text{PTS}}$
1 hr	72	64 8	1 0	2 8
	50	77 8	16 5	6 0
	69	85 5	41 5	16 0
	39	98 5	39 5	28 2
6 hr	52	4 0	3 0	0 0
	56	39 0	3 5	5 8
	74	57 8	23 5	8 4
	62	52 0	30 0	14 8

permanently injured sensory elements increases with the duration of the exposure A mechanical conception which corresponds to this view would be that on any portion of the basilar membrane a measure of its motion controls the probability of injury

A second view of the injurious action of the noise differs from the first in that the momentary probability of permanent injury to a sensory element depends on the element s history of exposure to noise According to this notion an element has more than the two states of being either normal or permanently injured A third state a susceptible state, is conceived One can imagine a *susceptible state of a* sensory element as either metabolic change with structure remaining normal or as an injury which would heal if further insult be prevented (It should be noted that the susceptible state could be represented as a series of sub lethal injuries graded from slight to severe) While in the susceptible state, a sensory element has an increased probability of being permanently injured Furthermore an element can return from the susceptible state to the normal state, that is an element can recover By these notions the proportion of elements permanently injured during a short interval will depend not only on the spectral and intensive properties of the noise, but also on the number of elements in the susceptible state The number of sensory elements in the susceptible state will in turn depend on the preceding *duration of a continuing exposure or on the opportunity for* recovery from a previous exposure to the noise By this conception the noise must do some work to get an element into a susceptible state and if recovery is allowed this work can be vitiated Our data seem to support this second conception of the process Nevertheless it may be that the first conception of the process is closer to the truth in the case of very intense sounds, that is it seems to us that extremely intense sounds should be able to permanently injure sensory elements immediately without an intermediate step of a susceptible state or of sub lethal injury

Protective mechanisms There is another possible explanation of the results of our experiment The cat can protect his inner ear from sound by closing his external ear and by contraction of his intra aural muscles as has been mentioned by Hawkins Lurie and Davis (1943) One might conjecture that *these protective mechanisms adapt during a continuous 2 hr exposure but* do not adapt nearly so much during a short $\frac{1}{8}$ hr exposure to the noise If the *recovery of the protective mechanisms* depends on the inter exposure interval then cats receiving widely spaced exposures would have more active protective mechanisms during a greater proportion of the on time of the noise than those cats receiving less widely spaced exposures Thus the more widely the exposures are spaced the lower the expected level of stimulation to the inner ear It seems to us that for the long silent intervals of the present experiment recovery of the protective mechanisms during the silent intervals would not be very different for the 1 hr 6 hr and 24 hr inter exposure intervals Therefore we believe that the role of protective mechanisms was *not* great in producing a difference among the three conditions for which the inter exposure interval was greater than zero It should be perfectly clear however that the difference between the continuous exposure and spaced exposures could be easily explained by the action of the protective mechanisms of the external and middle ear

VIII EFFECTS OF OCTAVE-BAND NOISE OF LOW FREQUENCY

The broad band noise of the previous experiments produced effects in the range from 125 to 500 cps which were surprisingly large. A mean PTS of about 32.0 db was found at 500 cps after the continuous exposures of 2 and 8 hrs. and an individual cat had a PTS as high as 61.0 db at this frequency. For these same exposures injury ratings for the apex and the upper part of Turn II attained mean values of 1.79 to 2.75 and individual ratings as high as 3.0 were noted for this portion of the basilar membrane. If the noise energy in the frequency region near 500 cps is largely responsible for these PTSs and injuries then it should be possible to produce them by exposure to an octave band of noise (300 600 cps) if the spectrum levels in the pass band match those of the broad band noise. The experiments of this section were designed to investigate this possibility.

As a first step in these experiments the masking pattern of the low frequency noise was measured. It is believed that these measurements give an estimate of the pattern of stimulation induced by the noise. After the masking data were obtained the cats were exposed to this low frequency band of noise and the post exposure threshold shifts were measured.

Subjects and Apparatus

The loudspeakers used to produce the deafening noise of the previous experiments see Chapter IV were placed in the audiometric booth. The speakers were aimed at the double grill cage. The exposures to the noise were administered in the double grill cage and not in the reverberant room. To produce the octave band noise the filtered output of a noise generator was amplified and placed across the speakers. An estimate of the spectrum produced in this way is shown in Fig. 11 of Chapter IV. This estimate was based on the following: (*a*) the assumption that the response of the loudspeaker was flat in this region, (*b*) the fact that the frequency response of the filters was measured and known, (*c*) the fact that overall level of the noise was measured at 16 positions distributed throughout the double grill cage. The median value of the overall SPL was taken as the SPL of this octave band of noise. The test tones were produced in the usual way.

Eight cats were used in these experiments. Four of these Nos. 53, 59, 61 and 70 had been used in an extensive study of masking of tones by noise reported by Watson (1963) and they had also undergone three brief exposures to the broad band noise. These previous exposures produced only TTS

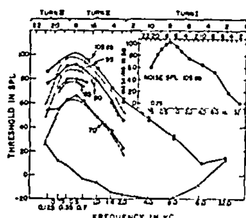


FIG 27 Masking pattern of a 300-600 cps band of thermal noise The ordinates are threshold values of the signal in SPL Along the top of the graph the distance along the cochlear duct is shown in mm from the round window Test tone frequencies are marked on the bottom of the chart at their probable locations of maximum stimulation The mean values of the masked thresholds are shown by the symbols connected by heavy straight lines The dotted lines show the estimated spectrum plus the signal to-noise ratio at masked threshold as measured by Watson (1953) The curve nearest the bottom of the graph shows the audibility curve for the cats of this experiment The inset shows the masking, the difference between the masked and quiet thresholds, for the 105-db SPL of the noise Each symbol on this figure represents the mean of measurements made on eight cats

The cats' audiograms were normal at the time of the present experiments The other four cats, Nos 75, 79, 98, and 99, had not been used in previous experiments

Masking

Masked thresholds in the presence of the band of noise (300-600 cps) were determined for each of four overall SPLs of the noise, 70, 85, 95, 105 db When the noise was at its highest level, 105 db, the limitations of the audiometer made it impossible to measure thresholds near the center of the noise-band, thus these thresholds had to be estimated from the masking within the pass band of the noise when the overall level of the noise was 95 db or less For this purpose it was assumed that the signal to noise ratio remains constant at the masked threshold When masked thresholds were measured at the 95 db and 105 db levels of the noise, only one measurement was made per cat per session, and for each cat the time between sessions was about 40 min In this way the possibility of injury being produced during the masking experiments was held extremely low All eight cats were used for the measurements

The results of this experiment are shown in Fig 27 On this figure the ordinates are the threshold values of the signal in SPL Along the top of the graph the distance along the cochlear duct is shown in millimeters from the round window On the bottom of the chart, frequencies are marked at their probable locations of maximum stimulation (Throughout this chapter the

anatomical frequency scale that was developed by Schuknecht (1953) is used) The mean masked thresholds are shown by the symbols connected by the heavy lines The dotted lines show the estimated spectrum plus the signal to noise ratio at masked threshold for a uniform noise measured by Watson (1963) The audibility curve for these cats is also shown The inset shows masking, the difference between the masked and the quiet thresholds for the noise at an SPL of 105 db

Please note that from 0 25 to 2 0 kc there is a close correspondence between the dotted lines and the solid lines Thus, over a broad range of frequencies the relative values of the masked thresholds may be anticipated from the knowledge of the spectrum and the signal to noise ratios of Watson s previous experiment

The discrepancies between the observed and predicted masked thresholds will now be discussed First consider the results for the frequencies near to or included in the pass band, 0 25, 0 35, 0 5, 0 7 kc In this case the 70 85 and 95 db levels produce about 2 to 4 db more masking than measured by Watson While the reason for this discrepancy is unknown it should be noted here that Watson measured signal to noise ratios in terms of voltages across the voice coils of the loudspeakers, since the same loudspeakers were used for tones and noise The signal to noise ratio of the present experiments depends on the acoustic calibrations of both the tones and the noise, since separate sources were used Certainly, in the present situation, these acoustic measurements are more subject to error than are the electrical measurements of Watson

For the test tone frequency of 125 cps an unexpected result was obtained The obtained masking was greater than the predicted amount by 27 db A listening test showed that hum or a resonance could not account for this 27 db discrepancy Also, additional threshold tests verified the values of masked thresholds This finding deserves further investigation It may be that this large amount of masking is similar to the remote masking of Bilger and Hirsh (1956)

The source of masking present at frequencies above 2 0 is unknown because neither the noise spectrum nor the response of the filter was examined above this frequency

The lowest curve of Fig 27 shows the quiet absolute thresholds for these cats The differences between the quiet and the masked thresholds are shown in the inset for the 105 db noise It can be seen that masking is produced over a broad range of frequencies and that the peak in the masking curve is at about 700 cps

Effects of Short and Prolonged Exposures to the Noise

All of the eight cats were exposed to the noise at the 105 db level for ½ hr Threshold tests were made at 0 35, 0 5, 1 0, 2 0, and 4 0 kc before and during the period from 17 to 42 minutes after the exposure The order of frequencies

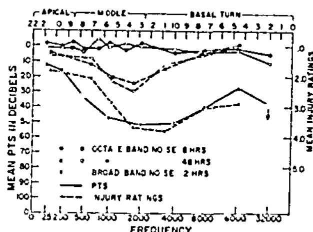


Fig. 28. Mean injury ratings and PTS after exposure to a low frequency band of noise (300-600 cps) for 8 or 48 hr. Data for an exposure to the broad band noise are shown for comparison.

was randomized. The mean TTSs were −0.62, −1.2, −0.62, 0.0, −1.87 from the low to high frequencies, respectively. Thus, no TTS could be measured at about ½ hr after the exposure.

The cats were then divided into two groups of four cats each. One group, the 8 hr group, was exposed to the 105 db noise for 8 hr, while the other group, the 48 hr group, was exposed to the same noise for 48 hr. As in the previous experiments designed to produce PTS, post-exposure testing followed the schedule given in Table 5.

Neither group suffered any significant PTS; that is, the threshold shifts of all cats were near zero when measured at 84 days after exposure. Injury ratings were done for these cats' cochleas even though no PTS had been measured. These ratings suggest that permanent injuries were present in the upper part of Turn I and the middle and lower parts of Turn II. These data are shown in Fig. 28; also shown are the comparable results for the 2 hr exposure to the broad band noise that was continuous in time. The symbols joined by solid lines show the PTS results, while those joined by the dashed lines show the mean injury ratings. From this figure it is obvious that even though the low frequency noise was on continuously for periods longer than the broad band noise by factors of 4 and 24, only negligible threshold shifts could be measured. The histological findings are, nonetheless, that slight injuries occurred in the upper part of Turn I and in the lower and middle portions of Turn II. These injuries are about the same as those noted after exposure to the broad band noise for ½ hour.

The location of the injury does not seem to be coincident with the mapped location of the peak of the noise, or the masking audiogram, or, as it will be shown, with the peaks of the TTS findings.

The TTS measured after the 8 and 48 hr exposures will now be discussed. Fig. 29 shows the TTS during the first few weeks after the 48 hr exposure. In panel A of this figure audiograms of individual cats are displayed. Note

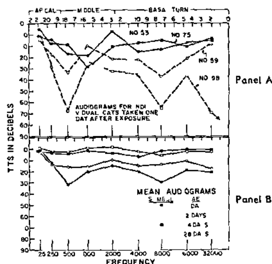


FIG 29 TTS after exposure to octave band noise (300–600 cps) for 48 hr The upper panel shows the audiograms of individual cats measured 24 hr after the cessation of the noise The lower panel shows the mean audiograms for these cats These audiograms were measured at 1 2 14 and 28 days after the cessation of the noise

that three of the four cats have audiograms with two peaks, that is, a low frequency dip between 0 25 and 1 0 kc and a high frequency dip between 4 0 and 16 0 kc are observed One of the cats, No 53, displays a single dip at about 1 kc Panel B shows the mean audiograms for this group at various times after exposure Note that both dips are retained as recovery proceeds

These results were not obtained after the 8 hr exposure In this case small shifts, usually less than 10 db, were measured one day after the exposure These small but definite shifts were spread throughout the frequency range By 7 days after the exposure these shifts were all within normal limits One exceptional result should be noted one day after exposure cat No 70 had a large TTS of 42 db at 8 0 kc This shift persisted through the second post exposure day, but by 7 days after exposure it had disappeared

Summary and Conclusions

In this set of experiments the masking pattern of an octave band of thermal noise (300–600 cps) was measured for an overall SPL of 105 db These results show that the relative values of the masked thresholds can be anticipated near and above the center frequency of the band, if the *spectrum of* the noise and the signal to noise ratio at masked threshold for tones at the center of a broad band of noise are known Nonetheless, unexpectedly large amounts of masking were measured at the lowest frequency of 125 cps

Exposure of the cats to this octave band noise for ½ hr produced no measurable effects on the threshold at about ½ hr after the exposure

Four of the cats were exposed to this noise for 8 hrs, while the remaining

four were exposed for 18 hrs. In most cases the 8 hr exposure produced slight threshold shifts (less than 10 db) throughout the frequency range. These threshold shifts persisted for about two days. The 18 hr exposure produced more marked threshold shifts (up to 68 db) which recovered over a period of about two weeks. Three of the four cats showed both a low frequency dip between 0.25 and 1.0 kc as well as a high frequency dip between 4.0 and 16.0. Neither exposure produced any PTSs which could be confidently accepted as greater than zero.

What appear to be significant injury ratings were noted for both the 8 and 18 hr exposure to this noise. These injuries were in the upper part of Turn I and in the middle and lower portions of Turn II which correspond to frequencies of about 1.0 and 2.0 kc.

Thus, masking has a peak about 700 cps, TTS is widespread with possible peak effects above and below 1.0 kc, PTS appears to be zero, and injuries were noted in a region of the basilar membrane corresponding to 1.0 and 2.0 kc. At the present time the relations among these variables are not understood. It may be that analysis like that of Tonndorf (1960) of the response of the basilar membrane to noise will aid in the clarification of these puzzling relations.

The octave band noise of these experiments and the broad band noise of the previous experiments showed nearly identical spectrum levels in the region from 300 to 600 cps. In this frequency range, however, no PTS was produced by the octave band noise, while large PTSs were measured after exposure to the broad band noise. Although the locations and magnitudes of the injury ratings are puzzling, those obtained after exposure to the octave band noise are very much less than those obtained after exposure to the broad band noise.

The differences between the effects produced by the octave band noise and those produced by the broad band noise could be attributed to many factors. For example, the exposure to the broad band noise was in a reverberant room while the exposure to the octave band noise was in a dead room; thus different directivity patterns existed. Or it could be argued that the particular frequency content of the band was the critical variable which produced the differences between the two experiments. While both of the above hypotheses have some merit, we believe that the results of this chapter suggest that, at least at low frequencies, PTS and injury are related to the bandwidth of the exposure stimulus. If future experiments confirm the importance of bandwidth, then it will be interesting to determine the relative importance of the associated variables of overall level and peak factor.

IX HISTOLOGICAL FINDINGS

By Walter P Covell

The Scale for Judging Degrees of Injuries

The injuries to the organ of Corti that were observable by our methods ranged from complete destruction to barely discernible alterations of the supporting and sensory cells The minimum amount of change that is recognizable by light microscopy and the usual hematoxylin and eosin stains consists primarily of a swelling of cytoplasm vacuolization, and minor displacements of cells It is extremely difficult to judge minor changes and it may be that further study by histochemical methods and electron microscopy will explain differences that now exist between minor injuries and the functional tests of the present data On the other hand it seems to us that the presence or absence of moderate to severe injuries can be determined with reasonable certainty by ordinary histological methods

The scale by which injuries were judged is described below Given in this description are those effects of intense sound which served as the principal criteria for each grade of injury There are other features of the inner ear which may influence the judging and these are discussed later

Grade 1 0 No discernible changes

Grade 1 5 Slight changes are apparent in supporting cells such as Deiters and internal phalangeal These are usually found to be cytoplasmic vacuoles or just distinguishable swelling

Grade 2 0 Mild to moderate changes become apparent in the supporting cells The nuclei may be enlarged and stain poorly or be slightly shrunken and the cytoplasmic changes more pronounced

Grade 2 5 Moderate to severe changes in supporting cells without appreciable injury to external hair cells The internal hair cell may be somewhat altered and the internal phalangeal markedly swollen A partial collapse of one of the tunnel rods is sometimes present

Grade 3 0 In addition to the changes listed above (Grade 2 5), external hair cell injury of slight degree is apparent This consists usually of cytoplasmic changes, but a deeply stained and shrunken nucleus or an enlarged and poorly stained nucleus may be present

Grade 3 5 Nuclear and cytoplasmic changes in Deiters cells become marked and occasionally these cells are loosened from the basilar membrane External hair cell changes are apparent to a moderate degree although the organ of Corti and its reticular lamina are not ruptured The tunnel of Corti may be almost completely collapsed

Grade 4 0 (See Fig 31) Supporting cells may show moderate to marked changes



Fig. 30. A mid-modiolar section through a cat's cochlea showing the different turns with the exception of the lower or basal portion of the first turn. T_{1M} middle of first turn; T_{1U} upper part of first turn; T_{2L}, T_{2M}, T_{2U} lower, middle and upper parts of second turn; T_A part of the small apical turn. ×12.

External hair cells are extremely altered in an otherwise intact organ of Corti; an occasional one may be missing. The reticular lamina may show a point of rupture.

Grade 4.5. This grade of injury is the greatest that can occur without complete rupture and loss of the organ of Corti from the basilar membrane.

Grade 5.0. The external hair cells and Deiters' cells have been lifted from the basilar membrane. Sometimes the internal hair cell and internal tunnel rod remain in a relatively unaltered state, as may also external and internal sulcus cells. This grade of injury also includes the complete loss of the organ of Corti from the basilar membrane.

The scale given above was used to rate each of seven regions of each cochlea. The histological methods for obtaining stained and mounted sections of the temporal bone were identical to those described for the guinea pigs by Covell and Eldredge (1951) except that the sections of the cat material were usually 18–20 microns in thickness. The approximate locations of the regions which were rated are shown in Fig. 30. The lower part of the basal turn does not appear in this mid-modiolar section. It should be recognized that these regions are approximate and that neighboring sections were often used to extend and confirm the observations made on the mid-modiolar sections.

It is worthwhile to compare the present scale for the cat with the previous scale developed by Davis and Associates (1953) for the guinea pig. The results for the guinea pig have been based largely on animals with severe trauma and short post-exposure lives. Discrepancies between this scale and



FIG 31 Lower part of the second turn of a mid modiolar section through the cochlea of a cat The reticular lamina is ruptured, external hair cells and supporting cells are markedly altered The internal hair and supporting cells show less marked changes There is some loss of mesothelial cells This is actually a severe grade of injury although the organ of Corti remains on the basilar membrane and the tunnel is intact except for fracture of the external tunnel rod There was degeneration of some peripheral nerve fibers and spiral ganglion cells from this area This was classified as a Grade 4 injury × 140

electrophysiological tests of function have been noted by Eldredge and Associates (1957, 1958, 1959, 1961) for certain exposure conditions, and examination of the present series of cats has suggested certain changes in the scale These amount to differences in the emphasis placed on the various signs of injury

Before discussion of these differences, it should be noted that the data provided by the cats cochleas differed in two important ways from those previously obtained from the guinea pigs cochleas

The cytoplasm of Deiters cells stains deeper for the cat than for the guinea pig Some of this may be due to the increase in thickness of the section It is therefore probable that minor changes in the cytoplasm of these cells are detected more readily in cat than in guinea pig

Unlike most of the guinea pig preparations, nearly all of the cats had long post exposure lives It was therefore, possible to see degeneration of spiral ganglion cells and of peripheral nerve fibers The presence of these degenerative changes was of value in confirming the importance of the extent and degree of changes in the organ of Corti They served as a basis for the modification and extension of the importance assigned to various injuries observed in other structures of the inner ear

The differences in the present scale of injuries and the previous scale for

guinea pigs are related primarily to the condition of Deiters' cells. A progression from mild changes in these supporting cells associated with normal hair cells to severe changes in both types of cells seems to have emerged. Accompanying changes in cell groups and membranes other than Deiters' cells, external hair cells and nerve cells have assumed a position of somewhat less importance. Certain of these features which were previously believed to be necessary for a moderate or severe grade of injury are commented on below.

Rupture of Reissner's membrane is not always consistent with other signs of injury and it has been observed to occur even with relatively mild changes in the organ of Corti. It should be remembered that even with careful examination of the appearance of the ruptured ends of a membrane, it is not always possible to differentiate ruptures produced by the sound from artifacts produced in the preparation of the sections. Stretching or rupture of one or both layers of Reissner's membrane does not correlate to any great degree with other signs of injury.

The loss of mesothelial cells is not always apparent even for severe grades of injury. Consistent changes in these cells have not been observed when slight and moderate degrees of injury are present to the organ of Corti.

The internal hair cell and its phalangeal cell may sometimes present few if any changes in regions showing marked injury to the external hair cells and Deiters' cells. They may show marked injury in regions where the external hair cells and Deiters' cells are relatively unchanged. In other words, the severity of injury to the internal hair cells cannot always be correlated with the grades of injury.

The tunnel of Corti in the presence of damaged supporting cells can undergo varying degrees of collapse, or it may remain fairly well intact as shown in Fig. 31. In the case of this sign of injury, as in the case of the other ancillary signs mentioned above, no precise relation between it and scale has been established.

Comments on Findings

In addition to the comments on the histological findings which are presented below, the following results are presented: the results of the judging of each cochlea are given in the table and the accompanying text of Appendix A; means and variances of the ratings for cochleas grouped by exposure conditions are given in the relevant portions of this monograph; and a comparison of behavioral and histological findings is given in Chapter X.

General. While different animals exposed to the same conditions often had widely different histological findings, the locations of the injuries along the basilar membrane appear to be nearly common to all: the upper part of Turn I and the region from the lower into the middle part of Turn II seem to be the areas of the cat's cochlea that are most susceptible to injury by these exposures. Nevertheless, it should be noted that after the longest expo

sures of 2 and 8 hr severe injury sometimes included the lower part of Turn I and, rarely, the injuries extended into the apical turn.

Comparison on the basis of duration of exposure. The broad band noise for ¼ hr produced changes in the cat's cochlea that were very slight. For the ½ hr exposure the injuries were mild in degree, and with the exception of one animal they were found to be greater than those found in the case of the ¼ hr exposure. The exposures of 2 hr duration produced moderate to severe injuries, and those for 8 hr resulted in severe injuries.

In the majority of the cochleas the lesions were more in evidence in the middle and upper portions of the first turn and in most of the second turn than in other portions of the cochlea. Approximately one half of those exposed for 2 hr also revealed moderate to severe injuries in the lower part of the first turn. A pattern similar to that found for the 2 hr exposures was also found for the 8 hr exposures. In the latter all but one cochlea showed moderate or severe injury in the lower portion of Turn I.

In general, it can be stated that the amount of injury increased with the duration of exposure to the broad band noise. The differences are greatest between the ¼ hr and the 2 hr exposures, although the injuries became slightly more severe when the duration of the exposure was increased from 2 to 8 hr.

Duration of post exposure life. A group of untrained cats was sacrificed two to four hours after exposure to the broad band noise at 115 db for 2 hr. The severity of the lesions was only slightly less, in most instances, than for the trained cats that had post exposure lives of 80 to 90 days. Fewer of the untrained cats showed a grade of injury in the lower part of Turn I comparable with that for the majority of the trained cats with long post exposure lives.

Spacing of exposures. Exposure to the broad band noise at 115 db in 16 doses of ⅛ hr each (two hours total) with an inter exposure interval (I.E.I.) of 1 hr resulted in slight to moderate changes in three and moderate to severe injuries in the fourth cat. When the experiment was repeated with a new set of four cats and the I.E.I. increased to 6 hr, comparatively slight injuries were observed.

If the injuries for the continuous, 2 hr exposures are compared with the spaced exposures with the same total duration, it is evident that the continuous exposures are more detrimental than the spaced exposures. These differences are more marked for the six hour interval between exposures than for the 1 hr interval.

Prolonged exposure to an octave band. A narrow band of low frequency noise (300–600 cps) at an overall SPL of 105 db resulted in slight injuries when the duration of the exposure was 8 hr, and it resulted in slight to moderate changes when the duration was 48 hr.

X RELATIONS BETWEEN TTS AUDIOGRAMS, PTS AUDIOGRAMS, $\overline{PTS}$, INJURY RATINGS, AND OTHER DEPENDENT VARIABLES

Introduction

In previous chapters the dependent variables have been grouped and discussed in relation to the parameters of the exposures. In the present chapter the data are organized by the severity of the average threshold shifts, specifically $\overline{PTS}$, and the major emphasis is placed on the analyses of the relations between dependent variables. This re-examination of the data was prompted by an inspection of the results for individual cats which suggested that knowledge of $\overline{PTS}$ specified the remaining dependent variables.

For most of the analyses to be presented in this chapter only data from cats exposed to the broad band noise are used; thus the spectrum of the noise is a constant and only the gross temporal characteristics of the noise are variable. That the spectrum was constant makes more plausible the necessary working assumptions that the relations between the dependent variables are largely independent of differences either in the exposures or in the susceptibility of the individual cats. In most instances the data do not seem to conflict with these assumptions.

In the course of the analyses of the relations among the dependent variables the characteristics of the PTS audiograms are described in detail. It is therefore convenient to include in this chapter a discussion of the important relations between PTS audiograms, the spectrum of the noise, TTS audiograms, and CTS audiograms.

$\overline{PTS}$ *and* PTS *Audiograms*

The degree to which knowledge of the value of $\overline{PTS}$ specifies a cat's PTS audiogram is examined below. In the course of this examination the frequency characteristics of the audiograms are described and inferences concerning the growth of PTS are made.

A PTS audiogram is shown in Fig. 32 for each of 34 cats that were exposed to the broad band noise. For this figure the audiograms have been ordered with respect to the value of $\overline{PTS}$ and the rank of the $\overline{PTS}$ for each audiogram is shown by the arabic numeral in the upper left hand corner of each panel. Also shown on each panel are the cat's number, the exposure condition, and the value of $\overline{PTS}$. On this figure C stands for continuous exposure while S stands for spaced exposures. The inter-exposure interval is II I. Most

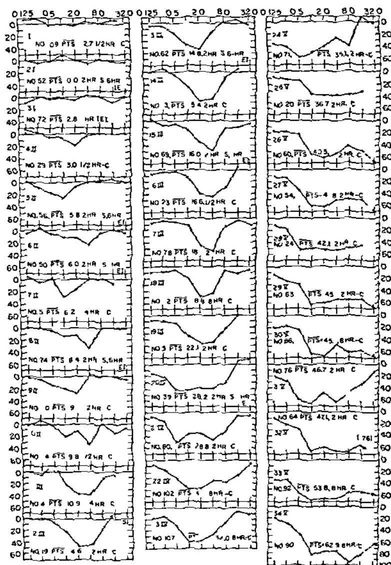


Fig 32 Audiograms showing persistent threshold shifts induced by exposure to noise for each of 34 cats All cats were exposed to the same broad band of thermal noise at an overall SPL of 115 db The temporal characteristics of the exposures did differ The grid marks on the axis of ordinates are 10-db steps and the grid marks on the axis of abscissas show the frequency of the test tones in kilocycles/sec See text and Appendix B for details

of these audiograms were measured 84 days after the cessation of the noise exposures Inspection of Fig 32 suggests that the audiograms, when ranked as they are by $\overline{PTS}$ form a graded series of increasing severity After examination of this series it was decided that the major trends shown by the audiograms were most easily described if the audiograms were categorized into five classes The class of each audiogram is given by the roman numeral

in the upper left hand corner of each panel of Fig 32 and the properties of each class are described below

The first three panels show the audiograms for the cats in Class I These audiograms are almost unaffected by the noise, and their $\overline{\text{PTS}}$s range from −2 7 to 2 8 db

Class II includes the seven cats shown on the panels ranked 4 through 10 The audiograms in this class have a dip at one of the octave steps in the mid frequency range 1 0 2 0, or 4 0 kc and $\overline{\text{PTS}}$ ranges from 3 0 to 9 8 db For two of the cats the dip is at 1 0 kc for two others the dip is at 2 0 kc and for the remaining three it is at 4 0 kc In all seven cases there is some PTS at 1 0 and 2 0 kc while only three have significant PTS at 4 0 kc A transition into the next category is represented by the audiogram for Cat No 114 shown in panel 10 in this case the dip is broadening to include a range of two octaves

The audiograms in Class III are shown in panels for ranks 11 through 19 for these nine cats $\overline{\text{PTS}}$ ranges from 10 9 to 22 0 db These audiograms are characterized by a dip that includes two of the octave steps The dip includes 1 0 and 2 0 kc *for one of the cats while for the remaining eight it includes* 2 0 and 4 0 kc

The next four panels ranks 20 to 23, show the audiograms of Class IV In this class $\overline{\text{PTS}}$ ranges from 28 0 to 32 0 db The audiograms are characterized by large shifts throughout three or four octaves of the mid frequency range In Class IV the shifts are not as great in either the low or the high frequency range as they are for the mid frequencies

The last eleven audiograms ranks 24 to 34 were all placed in Class V The $\overline{\text{PTS}}$s in Class V range from 35 0 to 62 9 db and the shapes of the audiograms are distinguished by severe shifts at either the high or the low frequencies as well as the mid frequencies Two of the cats present anomalous results The shape of Cat No 76 s audiogram rank 31 would place it in Class IV but the large value of $\overline{\text{PTS}}$ places it in Class V The histological findings for this animal are consistent with the large threshold shifts The *results for Cat 71 whose rank is 24 are difficult to interpret* This cat s audiogram has its largest shifts in the low and mid frequency range The interpretation of this finding is complicated by the facts that this cat had a slight high frequency loss before exposure to the noise that it was probably the oldest of the cats used in these experiments and surgical destruction of his left cochlea unlike all other cats in these experiments was not complete

The mean audiogram for each of the five classes of audiograms is shown in Fig 33 These mean audiograms reflect some of the trends noted in the series of individual cats In summary when $\overline{\text{PTS}}$ is less than about 3 db the deviations from the zero line appear to be random When $\overline{\text{PTS}}$ lies between 3 and 10 db a dip which encompasses about one octave is expected in the neighborhood of 1 0 or 2 0 or 4 0 kc A larger dip centered about 2 0 and 4 0 kc 1 0 and 2 0 kc can be expected when $\overline{\text{PTS}}$ lies between about 11 and 23 db For $\overline{\text{PTS}}$s in the range from about 24 to 35 db a dip with a

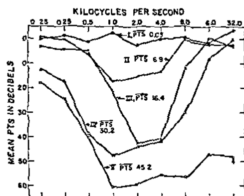


Fig 33

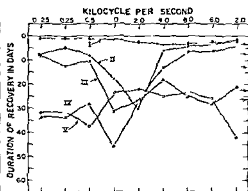


Fig 34

Fig 33 Audiograms showing the mean PTS for each of the five classes of severity On the figure the class is indicated as well as the mean value of $\overline{PTS}$

Fig 34 Mean duration of the recovery period for each frequency of the test tone The symbols on the far right of the figure are the mean number of days for the mean CTS $\overline{CTS}$ to recover within 6 db of the $\overline{PTS}$ For this analysis the cats were placed in classes of severity of $\overline{PTS}$

broad maximum which covers three or four octaves can be expected As $\overline{PTS}$ increases from 35 to 63 db, PTSs increase at all frequencies, but the greatest increases can be expected at the high frequencies

The data presented in Figs 32 and 33 support the hypothesis that knowledge of $\overline{PTS}$ specifies the PTS audiogram We further suggest that the sequence of increasing severity shown in Fig 32 may be taken as a picture of the development of PTS in an individual cat as the duration of the exposure is increased *It should be recognized that threshold shifts begin in* the neighborhood of either 1 0 or 2 0 or 4 0 kc The area of loss then spreads in the manner indicated by the series shown in the figure Furthermore we believe that if a new cat were given one of the exposures in these experiments, the best guess of the shape of his PTS audiogram would be made as follows The mean value of $\overline{PTS}$ for the group of cats previously exposed should be determined then from Fig 32 the audiogram of the cat with a value of $\overline{PTS}$ closest to the mean $\overline{PTS}$ should be taken as the predicted PTS audiogram An alternative procedure would be to use the mean audiogram for the group of cats which may include audiograms from several classes and thus not reflect the most probable shape of the audiogram for an individual cat

We also wish to comment here that the audiograms shown in Figs 32 and 33 support the common generalization that low frequencies are more resistant to acoustic trauma than are the mid and high frequencies, nonetheless the large PTSs of up to 35 db at 125 cps and 250 cps or up to 61 db at 500 cps should not be ignored In general it should be noted that PTSs in the octaves below 4 kc are more frequent and greater than the shifts in the two octaves above 4 kc For TTS audiograms produced by exposure to the spectrum

quite the opposite is true. This difference is discussed in more detail later in the chapter.

The audiogram categories represented by Classes I to V appear to be sufficiently homogeneous and significant to warrant examination of the mean values of other dependent variables for each class. In the sections that follow the division of the cats into Classes I to V is used for the examination of the period of recovery and the amount of recovery, for comparison of the behavioral and the histological findings, for comparison of the PTS audiograms with the spectrum of the noise, and for comparison of TTS, CTS, and PTS audiograms.

Period of Recovery

It will be recalled that the time required for the completion of recovery was measured as follows: the number of days required for the threshold shift to reach a value that was 6 db greater than the PTS was found by linear interpolation. Fig. 34 shows the mean number of days of recovery for each of the classes of severity. The cats in Class I had no PTS, and therefore the days to recover from TTS are shown. For this class recovery is complete at every frequency in less than four days. The curves for Classes II and III, however, show a strong frequency effect. The mid frequencies recover over a longer period of time than do either the low or the high frequencies. The duration of the recovery period differs little for Classes IV and V. For these classes there is a suggestion that the low frequencies require a little more time for recovery than do the high frequencies.

A source of bias in all of the curves of Fig. 34 is that a few instances of recovery from pure TTS are being averaged with the recovery of the temporary component of CTS. When the instances of TTS were eliminated from the data, the mean curves shown in Fig. 34 were not appreciably altered.

There is great variability in this measure of the duration of the recovery process, and therefore interpretations must be made with extreme caution. Nonetheless, it appears that the duration of the recovery period increases with increases in $\overline{PTS}$, at least up to values of 30 or 35 db for the latter variable. The symbols on the right hand margin of Fig. 34 show the mean number of days for the average threshold shift at the octave steps from 125 to 16 000 cps ($\overline{CTS}$) to reach a value within 6 db of $\overline{PTS}$. This parameter also increases as the mean $\overline{PTS}$ increases.

Amount of Recovery

In Table 12 the mean amount of recovery is shown for each of the five classes. These amounts of recovery were calculated by subtracting the PTS from the CTS at one day after exposure; thus, the amount of recovery is defined as the size of the temporary component of the compound threshold shift measured at one day after exposure. If the cat did not respond to the audiometer [illegible] output on the first post exposure day, the threshold

TABLE 12 *Amount of recovery (TS_1–TS_{81}) for each class of audiogram*

Audiogram class		Frequency in kc/sec 0.15	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	$\overline{PTS}$
I	$\bar{\chi}$	0.3	4.6	−0.1	9.7	7.5	13.3	14.1	14.5	13.2	7.1
	σ_m	2.9	4.2	3.4	2.2	3.3	10.2	4.5	4.8	4.7	3.3
II	$\bar{\chi}$	6.6	16.8	18.6	30.8	37.8	21.0	18.0	12.8	14.6	19.8
	σ_m	3.8	6.8	6.2	5.9	5.1	6.8	6.9	3.9	8.5	4.2
III	$\bar{\chi}$	19.4	31.6	45.4	43.4	22.9	25.0[a]	39.8[a]	21.1	28.4	31.9
	σ_m	9.5	10.9	14.9	11.3	5.7	11.8	19.9	6.9	14.2	10.5
IV	$\bar{\chi}$	40.2	47.0	47.4[a]	42.0[a]	45.1[a]	44.0[a]	48.9[a]	49.5[a]	56.1[a]	45.5[a]
	σ_m	4.3	3.5	11.1	10.3	12.1	8.2	9.4	13.7	7.1	7.8
V	$\bar{\chi}$	46.3[a]	52.1[a]	52.8[a]	42.5[a]	37.4[a]	38.1[a]	33.4[a]	29.5[a]	33.0[a]	41.4[a]
	σ_m	4.2	3.9	5.6	2.9	2.0	4.2	4.2	5.9	10.9	2.3

[a] Lower bound on sample mean

was taken as the maximum SPL of the audiometer, and a lower bound on the CTS was calculated. Mean values obtained using these lower bounds for individual cats represent lower bounds on the sample means. Many such instances can be noted in Table 12 and this fact degrades the usefulness of these particular measures. Nevertheless, it is clear that the amount of recovery —thus the temporary component of CTS—increases from Classes I to IV. Whether or not there is an increase in the amount of recovery as one goes from Class IV to Class V cannot be determined.

Injury Ratings and PTS

To facilitate the comparison of behavioral audiograms and injury ratings, a plot showing injury ratings as a function of frequency was devised. This was accomplished by converting the locations for which the ratings were made into frequency by means of the map of the cat's cochlea given by Schuknecht (1953). Thus a plot showing injury ratings in a manner which corresponds to the familiar audiogram was made. The panels of Fig. 35 show the mean injury ratings for each of the behaviorally defined classes of severity. Comparison of Figs. 33 and 35 shows a remarkable correspondence between the two sets of data. The only gross discrepancy between these behavioral and histological audiograms is for Classes I and II. The behavioral audiograms for these two classes are clearly different, while the mean injury ratings appear to be identical.

The relation between the behavioral threshold shift and the histological findings was examined in another way. In an attempt to use all of the data in evaluating this relation, $\overline{PTS}$ was correlated with the mean of the injury ratings along the length of the membrane. Fig. 36 shows the scattergram. On this graph each point shows the $\overline{PTS}$ and the mean injury rating ($\bar{I}$) for

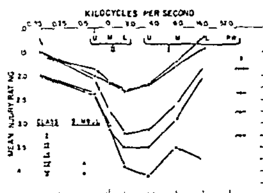


Fig. 35

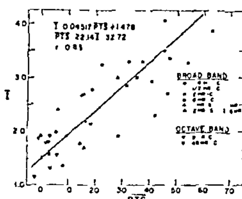


Fig. 36

Fig. 35. Mean injury ratings for the five classes of severity of $\overline{PTS}$. The ordinates are the mean injury ratings for the cats in each of the severity classes. The abscissas are locations along the basilar membrane. For convenience these positions are mapped to the test tone frequency that would give maximal stimulation at that point.

Fig. 36. Scattergram showing relation between the mean injury rating $\bar{I}$ and $\overline{PTS}$. Each point is for an individual cat. The ordinate is the mean of the injury ratings made at the several locations across the basilar membrane; the abscissas are $\overline{PTS}$s, means of the PTSs at the eight frequencies which comprise the octave steps from 125 to 16 000 cps.

a particular cat. For these correlations the cats exposed to the low frequency noise are included. The linear R for this relation is 0.83. The line shown in Fig. 36 is the least squares fit that minimizes the squared perpendicular distances between the points and the line. By this equation,

$$\overline{PTS} = 22.14\,\bar{I} - 32.72,$$

or

$$\bar{I} = 0.045\,\overline{PTS} + 1.48.$$

A similar plot was made for the mid frequency range. The average PTS at 1.0, 2.0, and 4.0 kc was compared with average injury rating in the upper part of Turn I and the lower and middle portions of Turn II. The linear R was 0.776 and the best fitting straight line was

$$PTS = 34.72\,\bar{I} - 68.47,$$

or

$$\bar{I} = .029\,PTS + 1.97.$$

By these equations $\overline{PTS}$ increases about 22 db for each unit increment to the average injury rating, while for the mid frequency range the corresponding slope is about 35 db. Zero $\overline{PTS}$ is associated with an average injury rating of 1.48, while for the mid frequencies zero PTS is associated with an average injury rating of 1.97. These intercepts reflect the fact that slight injuries were found in the middle of the basilar membrane when no threshold shifts were measured. Since all of these animals were exposed to noise, no control specimens were available; thus, this particular finding needs to be confirmed in experiments which include the appropriate control animals.

The results given above show, on the average, a close correspondence between the shapes of the behavioral audiograms and the pattern of injury ratings along the length of the basilar membrane. Furthermore, the average injury ratings for whole cochleas are highly correlated with $\overline{PTS}$. Nonetheless, many discrepancies between histological and functional measures remain. For example, it is clear from knowledge of the traveling wave in the cochlea that exact predictions of behavioral audiograms cannot be expected until rules are established for relating the extent of injuries along the basilar membrane as well as their severity to the threshold for a particular test frequency. In previous investigations by Eldredge and Associates (1957, 1958, 1959, and 1961) electrophysiological measures of cochlear function have been correlated with histological changes. These experiments suggest that the kinds of injuries observed histologically strongly depend on the sound pressure of the deafening stimulus, whereas equivalent functional changes are observed for both moderate and intense pressures if the duration of the exposure is appropriately adjusted. In general, a lowering of the correlation between any functional measures and histological measures can be expected if the spectrum of the deafening stimulus is allowed to vary within the series of experiments.

Comparison of TTS, CTS *and* PTS *Audiograms*

It was previously noted that after exposure to the spectrum of the present experiments the TTS audiograms peak at 4.0 kc. In addition, TTS spreads more to frequencies above than frequencies below 4.0 kc. Showing this relation quantitatively are 14 of 16 cats that had greater values of TTS at 8.0 kc than at 2.0 kc.

PTS audiograms after more severe exposures to the same spectrum show quite another picture. In this case the shifts are larger below 4.0 kc than they are above it. If one examines the audiograms in Classes II to V shown in Fig. 32 it can be seen that 28 of the 31 cats have greater values of PTS at 2.0 than at 8.0 kc. This is true even though for large $\overline{PTS}$ all mid- and high frequencies tend to have similar losses.

What is the shape of the CTS audiogram during the first hours after a severe exposure to the noise? Does it have the frequency characteristics typical of TTS audiograms, a TTS audiogram added to a PTS audiogram, or a PTS audiogram? Unfortunately it was often impossible to measure CTS audiograms during the first and second day after an exposure. Data that we were able to obtain during the first 6 to 48 hours after exposure suggest that CTS audiograms have the appearance characteristic of PTS audiograms. These limited data suggest that CTS audiograms form a series similar to that for PTS audiograms, that is, the shape of the CTS audiogram seems to depend on its average value at the time the audiogram was determined.

Shown in Fig. 37 are four examples of recovery of the temporary component of CTS audiograms. An example is taken from each of the classes

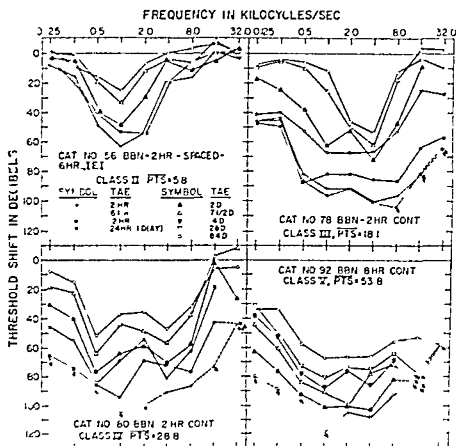


Fig. 37. The progressions from CTS audiograms measured shortly after exposure to the stable PTS audiogram. Changes in the shape of the audiograms as the temporary component of the CTS diminishes are exemplified by these four cats.

of severity. It can be seen that changes in the audiogram during recovery follow the general course of moving from severe to less severe along the graded series of audiograms like that previously shown in Fig. 32 and described in this chapter.

To summarize, if a cat is exposed to a spectrum for a duration only sufficient to produce TTS and, later, is exposed to the same spectrum for a duration sufficient to produce PTS, then the TTS and PTS audiograms can be expected to differ in both their contours and the locations of the maximum shifts. In addition, there will be no obvious correspondence between the temporary components of a CTS audiogram and a TTS audiogram even though both are produced by the same spectrum. These findings do *not* support the hypothesis that PTS produced by a traumatic noise is simply due to an extension of the processes responsible for large values of TTS; rather these findings suggest that both PTS and the temporary component of CTS, when they are produced by traumatic exposures to sound, depend on processes different from those that are responsible for TTS.

Relation of PTS *Audiograms to the Spectrum*

One explanation of the difference between ITS and PTS audiograms is that they depend on different measures of the spectrum of the exposure stimulus. A simple hypothesis of this type is that the PTSs at the various test frequencies are proportional to the levels of the noise in narrow bands centered about each test frequency while ITSs are proportional to the levels in noise bands of lower frequency than the test stimulus. One might add in both cases that the spectrum levels should be corrected by the transmission characteristics of the ear and that these characteristics can be approximated by including the audibility curve or the critical ratio curve or both in such a formulation.

In this section we wish to evaluate a simple form of the hypothesis that PTSs are proportional to the spectrum levels of the exposure stimulus at the corresponding frequencies. This hypothesis is described as the spectrum matching hypothesis. It states that where significant permanent threshold shifts are present, the *post exposure audibility curve* can be matched by the spectrum of the noise minus a constant. For this form of the hypothesis, the additive constant is determined by the duration of the exposure and by a cat's susceptibility to the noise. That is, both individual differences and the duration of the exposure are treated as additive factors which are constant across test tone frequencies. This simple form of the spectrum matching hypothesis can be rejected for experiments which vary the bandwidth of the spectrum as shown by the comparison of narrow and broad band exposures in Chapter VIII of this monograph. The possibility remains that in the case of the broad band noise a strong correlation existed between the spectrum levels and the post exposure thresholds. This possibility was tested in the following way.

At those frequencies for which PTS was greater than 5.0 db, the difference between the spectrum level of the noise and the post exposure threshold was calculated for each of the 34 cats exposed to the broad band noise. The mean of these differences, C, was calculated for each cat. Since the PTS audiogram is the difference between the pre- and post exposure audibility curves, the spectrum matching hypothesis was evaluated by fitting the PTS audiograms in accordance with the equation

$$\text{PTS} = (B - C) - \beta_0$$

and if $(B - C) - \beta_0 < 0$

then PTS $= 0$.

PTS is the fitted value of PTS, B is the spectrum level of the noise at the appropriate frequency, β_0 is the SPL of the threshold stimulus before exposure to the noise. The constant C has a single value for each cat. Of course, the quantity $(B - C)$ is the fitted post exposure threshold.

To evaluate this form of the spectrum matching hypothesis, the correspon

TABLE 13 *Correspondence between observed PTS and PTS fitted to the data on the basis of a spectrum matching hypothesis*

Class		Frequency in kc/sec									
		0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	PTS
I	PTS	0.0	1.2	1.1	−2.4	2.8	0.0	0.4	1.5	3.2	0.03
	PTS	0.0	2.9	0.4	4.5	4.1	0.0	0.4			
II	PTS	0.4	0.6	4.4	17.3	15.6	13.3	−0.4	2.6	2.3	6.9
	PTS	0.6	0.0	4.9	13.5	13.6	5.8	−3.2			
III	PTS	3.0	4.6	5.0	22.0	42.9	40.8	11.9	0.2	2.6	16.4
	PTS	−0.2	5.0	17.6	32.0	37.0	29.8	20.4			
IV	PTS	12.5	17.2	38.6	47.5	44.2	42.0	30.2	8.0	0.5	30.0
	PTS	0.0	13.3	37.1	47.3	54.4	43.7	31.2			
V	PTS	17.9	21.5	29.9	60.7	59.5	55.3	56.4	47.1	>48.6	[illegible]
	PTS	4.7	32.3	19.0	61.3	65.5	55.4	50.6			

dence between PTS and PTS was examined for each cat. In some cases the fit was excellent while in others it was poor. The test frequency showing maximum PTS was not always predicted. Overall the root mean square of differences between the predicted and the obtained PTSs was about 9.6 db and the average difference was 8.0 db. Table 13 shows the correspondence between PTS and PTS for the five classes of audiograms. The agreement is generally fair except for Class III where it is poor.

Calculations similar to those reported above were made using the estimated masked thresholds in place of the spectrum level of the noise. This change in effect includes both the audibility curve and the critical ratio curve determined for the cat by Watson (1963) as corrections on the spectrum level. The fitted PTS audiograms obtained in this manner were no better or worse than those previously obtained.

We believe that the simple spectrum matching hypothesis presented above fails for several reasons. It does not include changes in the spectrum produced by the intra-aural muscles. It assumes that noise bands are mapped onto the basilar membrane in the same positions as the corresponding pure tones, which according to Tonndorf (1960) is incorrect. The assumptions that the constant of proportionality between spectrum level and PTS has unit value and that it is independent of exposure duration need investigation.

It should be possible to specify precisely the relation between the spectrum and PTS if data are accumulated which are free from the unknown effects of the intra-aural muscles. Information which would help bridge the gap from the pattern of the spectrum to the PTS audiogram could be obtained from experiments performed on either the cochlea or models of it that determine the relations among the measures of the motion of the basilar membrane and the severity of traumatic injury.

Further Comments on the Relations Between TTS, CTS, *and* PTS

For man, after exposure to intense transients or to most noises of broad bandwidth TTS, CTS, and PTS all seem to show a characteristic maximum near 4 kc for both traumatic exposures of short duration and high intensity and for long term exposures to less severe levels. There are instances, however, of such severe exposures that produce either PTSs or very severe TTSs which seem to have the frequency characteristics of a cat's PTS audiogram. For cat, it has been shown by the data of Lindquist, Neff and Schuknecht (1954), of Schuknecht, Neff, and Perlman (1951) and of the present report that exposures to gunshot or broad band noise, or blows to the head produce PTS audiograms that in most instances show more loss below 4 kc than above it. The opposite is true for the cat's TTS audiograms.

To account for these findings we suggest the following hypotheses.

1. Threshold shifts that are due to underlying temporary and permanent injuries produced by traumatic exposures of relatively short duration (order of seconds and hours) are proportional to the spectrum levels of the traumatic noise after its transformation by the mechanical mechanisms of the ear. One property of this hypothesized relation is that the maximum of the transformed spectrum should be the point of maximum PTS.

2. *Threshold shifts that are due to fatigue like processes also have a simple* relation to the spectrum of the exposure stimulus. This relation, however, involves some parameter which increases with frequency, thus moving the location of maximum threshold shift to a frequency higher than the maximum of the transformed spectrum. A TTS experiment which illustrates this position was conducted by Miller (1958).

3. Continued or repeated severe *fatiguing* of the ear over a period of many years produces PTS audiograms with the frequency characteristics of TTS audiograms. This kind of PTS corresponds to the noise induced threshold shift described by Davis (1960). A recent study by Nixon and Glorig (1961) casts doubt on this hypothesis. They examined the growth of PTS corrected by presbycusis at both 2.0 kc and 4.0 kc in men exposed to industrial noise over periods of many years. The spectra of the noises were such that short exposures (8 hr) to them probably produced TTS audiograms with maximums near 4 kc and little or no TTS at 2.0 kc. The spectrum levels nonetheless were maximum between 0.3 and 0.6 kc. The overall levels of these noises were such that PTS accumulates slowly over periods of years. The data of Nixon and Glorig show that initially PTS is greatest at 4.0 kc and it appears to reach an asymptotic level after ten years of exposure. On the other hand the PTS at 2.0 kc shows little or no growth during the first ten years of exposure. These data for the first ten years of exposure are consistent with the hypothesis that TTS audiograms and PTS audiograms have the same shape for noise induced deafness. During the period beyond ten years, however, continued exposure to the most intense noise of the Nixon and Glorig study produces marked increases in the PTS at 2.0 kc even though

the PTSs at 4.0 kc remain constant. Thus the final shape of the PTS audiogram is probably different from the shape of the TTS audiogram, the losses being greater in the range important for speech perception in the case of PTS than in the case of TTS.

4. The hypotheses stated above are subject to modification by consideration of the intra-aural muscles, which provide an unknown and changing frequency distortion of the exposure stimulus, and species differences which include structural and acoustical differences in all parts of the ear.

XI SUMMARY

Aural effects of exposure to intense noise were investigated by behavioral measurements of the auditory sensitivity of cats before and after such exposures and by histological examination of their cochleas

Procedures for the training and the audiometric testing of the cat are described in detail The results of extensive determinations of the cat s audibility curve for sound fields are reported and a standard audibility curve is offered for the use of future investigators

Terms that are useful in the description of the deafening effects of exposure to intense sound are defined as follows A threshold shift is computed as the difference in decibels between pre and post exposure thresholds Threshold shifts that are measured at post exposure times that exceed a few seconds and that decline in time to zero are denoted as temporary threshold shifts (TTSs) If after exposure to intense sound threshold shifts are measured that are stable and persist over a period of several weeks then these threshold shifts are said to be persistent threshold shifts (PTSs) and permanent injury to the auditory mechanism is inferred *Threshold shifts that have both temporary and fresh* persistent components are defined as compound threshold shifts (CTSs) Average values of CTS or PTS over the frequency range from 125 to 16 000 cps are denoted in this monograph by the symbols $\overline{CTS}$ and $\overline{PTS}$

The broad band noise that was used as an exposure stimulus is described in detail The noise had nearly equal octave band levels in the bands centered at 850 1700 and 3400 cps with lower levels above and below this region

Exposure of the cat to this noise either at an overall SPL of 115 db for ⅛ hr or at 105 db for ¼ hr results in TTS audiograms that have the same general features as those measured in man the maximum TTS is at 4 kc and TTS is greater above 4 kc than below it Recovery from large values of TTS is similar to that for man The cat is more susceptible than man to TTS it is estimated that if man and cat are to have equal TTS then the exposure must be approximately 18 db greater in energy for man than for cat

Cats also were exposed to the broad band noise at an overall SPL of 115 db without interruption for periods of ¼ ½ 2 or 8 hrs These exposures produced PTSs whose magnitudes depended on the duration of the exposure the test tone frequency and the susceptibility of the individual cat Mean values of $\overline{PTS}$ *were 5 6 db and 8 5 db for the ¼ hr and ½ hr exposures* while values of 35 0 db and 40 6 db were found for the 2 hr and 8 hr exposures The growth of PTS with increasing duration of exposure is described in detail in the text

6 6 901 Mdl

The effects of distributing the 2 hr exposure into 16 doses of ⅛ hr each were investigated. A different inter-exposure interval was used in each of four conditions; these were 0, 1, 6, or 24 hr. Average values of $\overline{PTS}$ were 38, 13, 6, and 2 db, respectively. Thus the greater the inter-exposure interval the smaller the PTSs. If PTSs are to be reduced to near zero values for all cats, a small duty cycle of 1.0 to 0.5% seems to be required for exposure schedules similar to those used in these experiments.

The behavior of the temporary component of CTS was examined for cats exposed to the broad band noise. The following conclusions are limited by the facts that it was often impossible to measure threshold shifts during the first few days after exposure and that PTSs exceeded 70 db in only one cat. The size of the temporary component at 24 hr after exposure (the difference between CTS at one day after exposure and PTS) increased with the severity of the exposure, and it was positively correlated with PTS. Recovery from the temporary component of CTS is most rapid during the first few post-exposure days; thereafter, the rate of recovery gradually slows. The period of recovery increases with the severity of the exposure, and it is also positively correlated with the magnitude of the PTS. This period ranged from a few days up to approximately two months. The recovery processes seem to have stopped after the first two post-exposure months.

The PTS audiograms produced by either continuous or spaced exposures to the broad band noise had characteristic shapes. For small average values of PTS, the PTS audiogram has a dip which includes one octave in the neighborhood of 1.0 or 2.0 or 4.0 kc, depending on the cat. As the average value of PTS increases, the dip widens to include first two and then three or four octaves of the range from 500 to 8,000 cps. For average values of $\overline{PTS}$ greater than 35 db, sizable PTSs can be expected at all frequencies, the largest usually being at frequencies above 250 cps. The shapes of CTS audiograms seem to depend on their average values in a manner similar to that described for PTS audiograms.

PTS and CTS audiograms produced by exposure to the broad band noise have greater shifts below 4.0 kc than above it, while the opposite is true for TTS audiograms produced by the same spectrum. Thus, PTSs and CTSs produced by traumatic exposure to sound seem to depend on underlying processes which differ from those responsible for TTS.

The auditory effects of an octave band of noise (300–600 cps) were also investigated using the cat as a subject. A band pressure level of 105 db was used since it matched the level of the corresponding band of the broad band noise whose overall level was 115 db. The masking pattern of the octave band was determined and this pattern was as expected except for unexplainably large amounts of masking found at 125 cps. Exposure to the octave band for 8 hr produced small but definite threshold shifts throughout the frequency range, and these shifts declined to near zero values by the seventh post-exposure day. Exposure to this noise for 48 hr produced TTS audiograms that had two dips, one between 0.25 and 1.0 kc and the other between

4 0 and 16 0 kc No PTSs were measured, were noted in the histological findings Fu of prolonged continuous exposures to low f the present results suggest that some variable miner of PTS and injury, at least for low fre

A scale for the histological evaluation of this scale injuries were rated at seven locatio of injuries from slight changes in Deiters Deiters and external hair cells appears t injury ratings along the basilar membrane the behavioral audiograms, if both are pla scale The Pearson correlation coefficient the whole cochlea and the cat s average PT

The data available for man show that the no persistent threshold shifts if exposed to in the present experiments We hypothesiz increased by approximately 18 db, then for the cat would be found in man

REFERENCES

Anthony, A., Ackerman, E., and Lloyd, J. A., 1959. Noise stress in laboratory rodents. I. Behavioral and endocrine responses of mice, rats, and guinea pigs. *J. Acoust. Soc. Am.*, *31*, 1430–1437.

Bilger, R. C., and Hirsh, I. J., 1956. Masking of tones by bands of noise. *J. Acoust. Soc. Am.*, *28*, 623–630.

Corso, J. F., 1958. Proposed laboratory standard of normal hearing. *J. Acoust. Am.*, *30*, 14–23.

Covell, W. P., and Eldredge, D. H., 1951. Injury to animal ears by intense sound. AF Tech. Rpt. No. 6561, Part I, Wright Air Development Center, Wright-Patterson AFB, Ohio.

Culler, E., Finch, G., Girden, E., and Brogden, W., 1935. Measurements of acuity by the conditioned response technique. *J. Gen. Psychol.*, *4*, 223–227.

Davis, H., 1960. In *Hearing and Deafness* (H. Davis and S. R. Silverman, Ed.). New York: Holt, Rinehart, and Winston, Inc., pp. 104–107.

Davis, H., Morgan, C. T., Hawkins, J. E., Galambos, R., and Smith, F. W., 1950. Temporary deafness following exposure to loud tones and noise. *Acta Oto-Laryngol.*, Suppl. 88. 57 pp.

Davis, H., and associates, 1953. Acoustic trauma in the guinea pig. *J. Acoust. Soc. Am.*, *25*, 1180–1189.

Dworkin, D., Katzman, J., Hutchinson, G. A., and McCabe, J. R., 1940. Hearing acuity of animals as measured by conditioning methods. *J. exp. Psychol.*, *26*, 281–298.

Egan, J. P., Greenberg, G. Z., and Schulman, A. I., 1961. Interval of time uncertainty in auditory detection. *J. Acoust. Soc. Am.*, *33*, 771–778.

Eldredge, D. H., Covell, W. P., and Davis, H., 1957. Recovery from acoustic trauma in the guinea pig. *Laryngoscope*, *67*, 66–84.

Eldredge, D. H., and Covell, W. P., 1958. A laboratory method for the study of acoustic trauma. *Laryngoscope*, *68*, 465–477.

Eldredge, D. H., Covell, W. P., and Gannon, R. P., 1959. Acoustic trauma following intermittent exposure to tones. *Ann. Otol. Rhinol. Laryngol.*, *68*, 723–733.

Eldredge, D. H., Bilger, R. C., Davis, H., and Covell, W. P., 1961. Factor analyses of cochlear injuries and changes in electrophysiological potentials following acoustic trauma in the guinea pig. *J. Acoust. Soc. Am.*, *33*, 152–159.

Elliot, D. N., Stein, L., and Harrison, M. J., 1960. Determination of absolute intensity thresholds and frequency difference thresholds in cats. *J. Acoust. Soc. Am.*, *32*, 380–384.

Elliot, D. N., 1960. Personal communication.

Galambos, R., and Rupert, A., 1959. Action of the middle ear muscles in normal cats. *J. Acoust. Soc. Am.*, *31*, 349–355.

Greenwood, D. D., 1961. Critical bandwidth and the frequency coordinates of the basilar membrane. *J. Acoust. Soc. Am.*, *33*, 1344–1362.

Hawkins, J. E., Jr., Lurie, M. H., and Davis, H., 1943. Supplementary report on injury of the inner ear by exposure to loud tones. OSRD Report, December 31, 15 pp.

Kryter, K. D., and Ades, H. W., 1943. Studies on the function of the higher acoustic nervous centers in the cat. *Amer. J. Psychol.*, *56*, 501–536.

Lindquist, S. E., Neff, W. D., and Schuknecht, H. F., 1954. Stimulation deafness: a study of hearing losses resulting from noise or blast impulses. *J. comp. physiol. Psych.*, *47*, 406–411.

Loeb, M., and Riopelle, A. J., 1960. Influence of loud contralateral stimulation on the threshold and perceived loudness of low frequency tones. *J. Acoust. Soc. Am.*, *32*, 602–610.

Miller, J. D., 1958. Temporary threshold shift and masking for noise of uniform spectrum level. *J. Acoust. Soc. Am.*, *30*, 517–522.

Miller, J D, Anderson, C. D, Gundy, R F, Watson, C. S, and Egan, J P, 1958 Interim Report for Contract AF 33 (616)–3844 Mimeograph report dated 26 March, Indiana University, Bloomington, Indiana

McGill, T E, 1959 Auditory sensitivity and the magnitude of the cochlear potential *Ann Otol Rhinol and Laryngol*, *68*, 193–208

Neff, W D, 1947 The effects of partial section of the auditory nerve *J Comp physiol Psychol*, *40*, 203–215

Neff, W D, and Hind, J E, 1955 Auditory thresholds of the cat *J Acoust Soc Am*, *27*, 480–483

Nixon, J C, and Glorig, A, 1961 Noise-induced permanent threshold shift at 2000 cps and 4000 cps *J Acoust Soc Am*, *33*, 904–908

Rudmose, W, 1957 Hearing loss resulting from noise exposure In *Handbook of Noise Control* (C M Harris, Ed) New York McGraw-Hill Book Co, Inc, Chapt 7 22 pp

Schuknecht, H F, 1950 A clinical study of auditory damage following blows to the head *Ann Otol Rhinol and Laryngol*, *59*, 331–338

Schuknecht, H F, 1953. Techniques for study of cochlear function and pathology in experimental animals A M A *Arch Otolaryngol*, *58*, 377–397

Schuknecht, H F, Neff, W D, and Perlman, H B, 1951 An experimental study of auditory damage following blows to the head *Ann Otol Rhinol and Laryngol*, *60* 273–289

von Schulthess, G Rechberg 1961 *Innenohr und Trauma* Basel Sokorger, 28–41

Simmons, B, 1959 Middle ear muscle activity at moderate sound levels *Ann Otol Rhinol and Laryngol*, *68*, 1126–1143

Sivian, L J, and White, S C, 1933. On minimum audible sound fields *J Acoust Soc Am*, *4*, 288–321

Swets, J A, 1961 Is there a sensory threshold? *Science*, *134* 168–177

Tonndorf, J, 1960 Response of cochlear models to aperiodic signals and to random noise *J Acoust Soc Am*, *32* 1344 1355

Ward, W D, Glorig, A, and Sklar, D L, 1958 Dependence of temporary threshold shift at 4 kc on intensity and time *J Acoust Soc Am* *30*, 944 954

Ward, W D Glorig, A, and Sklar, D L, 1959*a* Temporary threshold shift from octave-band noise applications to damage risk criteria *J Acoust Soc Am* *31* 522–528

— 1959*b* Relation between recovery from temporary threshold shift and duration of exposure *J Acoust Soc Am*, *31* 600–602

— 1959*c* Temporary threshold shift produced by intermittent exposure to noise *J Acoust Soc Am* *31*, 791–794

Ward, W D 1960 Recovery from high values of temporary threshold shift *J Acoust Soc Am*, *32*, 497–500

Watson, C S, 1963 Masking of tones by noise for the cat *J Acoust Soc Am*, *35* No 2 (*In press*)

APPENDIX A

Table showing grades of injury for each specimen
and an explanation of the table

APPENDIX A. *Grades of injury*

Cat no	Exposure	Turn I apex	Turn II upper	Turn II middle	Turn II lower	Turn I upper	Turn I middle	Turn I lower
Trained								
1	¼ hr b b n , c.,	1.5	2.5	3.0	2.5	2.5	2.5	1.5
5	115 db	1.5	1.5	2.5	2.0	1.5	2.0	1.5
Trained								
19	½ hr	1.5	2.0	2.0	3.0	4.0	4.0	3.0
23	b b n	1.5	1.5	2.5	3.0	3.5	3.0	2.0
29	c.	1.0	1.5	1.5	2.0	2.5	1.5	1.5
109	115 db	1.5	2.0	2.5	2.0	2.0	1.5	1.0
110		2.0	2.0	2.0	2.5	2.5	2.0	1.5
114		1.5	2.0	2.5	2.0	2.0	2.0	1.5
Trained								
3	2 hr	1.5	2.0	3.0	4.5	5.0	4.5	3.0
13	b b n	1.5	2.0	2.5	4.0	4.5	3.0	2.0
20	c.	2.0	2.5	3.0	4.0	4.0	3.5	3.5
24	115 db	2.5	2.5	3.0	4.5	5.0	3.5	3.0
54		1.5	1.5	2.0	3.0	2.5	2.0	3.5
60		1.5	1.5	2.0	2.5	4.5	4.0	4.5
63		2.0	2.5	4.0	4.0	3.5	4.0	4.5
64		2.5	3.0	3.5	4.0	3.5	3.0	4.0
71		1.5	2.5	3.0	4.0	4.5	3.5	2.0
76		2.0	2.0	3.0	4.5	4.0	2.0	1.5
78		1.5	2.0	3.5	3.0	2.0	1.5	1.5
80		1.5	1.5	2.0	2.0	3.0	2.0	1.5
Untrained								
A r	2 hr	1.5	1.5	2.0	3.0	2.5	2.0	1.5
l	b b n	2.0	2.0	2.5	3.0	2.5	2.0	1.0
D r	c	2.0	2.5	2.5	3.5	3.0	3.5	2.0
l	115 db	1.5	1.5	2.0	3.0	2.0	2.0	1.5
E r		1.0	1.5	2.0	3.5	2.5	2.0	1.5
l		2.0	2.0	3.0	3.5	2.5	2.0	1.5
F r		1.5	2.0	2.5	3.0	2.0	1.5	1.5
l		1.5	2.0	2.0	3.0	4.0	5.0	3.0
G.r		2.0	2.5	3.0	3.5	3.5	4.0	1.5
l		2.0	2.5	3.5	3.5	4.0	4.0	2.0
Trained								
86	8 hr	3.0	3.0	4.0	5.0	4.5	4.0	5.0
90	b b n	2.0	3.0	3.5	4.0	4.5	5.0	5.0
92	c	1.5	2.0	2.5	3.5	4.5	4.0	5.0
102	115 db	2.0	2.5	3.5	4.0	3.0	3.0	1.0
107		2.5	3.0	3.0	4.0	4.0	3.5	3.0
112		2.0	3.0	3.5	4.0	3.0	2.5	1.5
Trained								
39	2 hr b b n	2.0	2.5	4.0	4.0	3.5	3.0	2.0
50	c 10 doses	1.5	2.5	3.0	3.5	3.0	2.0	1.5

Appendix A (cont)

Cat no	Exposure	Turn I apex	Turn II upper	Turn II middle	Turn II lower	Turn I upper	Turn I middle	Turn I lower
69	7½ min	1 5	1 5	2 0	2 5	2 0	1 5	1 0
	ILI – 1 hr							
72	115 db	1 5	2 0	2 0	3 0	2 0	1 5	1 0
Trained								
52	2 hr	1 5	2 0	2 0	2 0	2 5	2 0	1 5
	b b n							
56	s	2 0	2 0	2 5	2 5	2 0	1 5	1 5
	16 doses							
62	7½ min	1 5	2 0	2 5	2 5	1 5	1 5	1 0
	ILI – 6 hr							
71	115 db	1 0	1 5	1 0	1 5	2 0	1 5	1 0
Trained								
61	8 hr	1 0	1 0	1 5	2 0	1 5	1 5	1 0
70	n b n	1 0	2 0	2 0	2 0	1 5	1 0	1 0
79	c	1 5	1 5	2 5	2 5	2 0	1 5	1 0
99	105 db	1 5	2 0	2 5	2 5	2 0	1 5	1 0
Trained								
53	48 hr	1 5	1 5	2 5	3 5	2 0	1 5	1 0
59	n b n	1 0	1 0	1 5	1.5	1 0	1 0	1 0
75	c	1 5	1 5	2 5	2 0	1 5	1 0	1 0
98	105 db	1 5	1 5	2 0	3 0	2 0	1 5	1 0

Abbreviations c, continuous s, spaced b b n, broad band of noise, n b n, narrow band of noise (300–600 cps) I L I, inter exposure interval

EXPLANATION OF TABLE

Trained cats 4 and 5 *Exposure* A broad band of continuous noise at 115 db for one quarter hour *Results* Usually mild injury to supporting cells

Trained cats 19, 23, 29, 109, 110, and 114 *Exposure* a broad band of continuous noise at 115 db for one half hour *Results* Mild injury to supporting cells with some involvements of external hair cells particularly in two specimens

Trained cats 3, 13, 20, 24, 54, 64, 71, 80, 60 63, 76 and 78 *Exposure* A broad band of continuous noise at 115 db for two hours *Results* Injuries in lower part of the first turn for four specimens show loss and degeneration of external and internal hair cells and supporting cells with collapse of the tunnel of Corti The similar areas in four other specimens reveal slight to moderate changes such as swelling of supporting cells with or without some deformation, of external hair cells In the upper part of the first turn and into the middle of the second turn, the damage is in fair agreement for most specimens

Untrained cats, A, D E, F, and G *Exposure* A broad band of continuous noise at 115 db for two hours Animals sacrificed within two to four hours following exposure Both ears were exposed *Results* Only relatively mild changes are usually present in the lower part of the first turn, such as slight swelling of supporting cells These changes become somewhat greater in the middle and upper part of the first turn and for three specimens severe injuries are present in this location Definite hair cell and other cell injuries of moderate degree are in evidence in the lower part of the second turn for all specimens These become less in the middle and upper part of the second turn

Trained cats 86 90 92 102, 107 and 112 *Exposure* A broad band of continuous noise at 115 db for eight hours *Results* In three cochleas the organ of Corti was lifted from the basilar membrane in the lower part of the first turn In one there was moderate injury and in two, slight changes at the same location For all specimens the damage became more severe or maintained its severity into the middle of the second turn For the remainder of the apical turn the injuries were moderate to slight

Trained cats 39 50 69 and 72 *Exposure* Sixteen doses, 7 5 minutes each (or two hours total) with one hour interexposure interval *Results* The extreme ends of the basal and apical turns reveal only slight or mild injuries such as supporting cell changes In the upper part of the first turn and into the middle of the second turn there is considerable variation in degree of

injury for the different specimens. It ranges from supporting cell changes to marked organ of Corti damage for one specimen.

Trained cats 52, 56, 62 and 74. *Exposure:* Sixteen doses, 7.5 minutes each (or two hours total) with six hour interexposure interval. *Results:* The injuries for all specimens are slight or relatively mild. There is a tendency for the greatest degree of injury to occur in the upper part of the first turn and lower and middle parts of the second turn. It is primarily supporting cell changes with some evidence of slightly altered hair cells.

Trained cats 61, 70, 79 and 99. *Exposure:* A narrow band of low frequency (300–600 cps) continuous noise at 105 db for eight hours. *Results:* Very mild injuries are present chiefly in the upper part of the first turn and throughout the second turn for each specimen. These are primarily supporting cell changes with slight, if any, hair cell damage. As a group, the findings are fairly consistent.

Trained cats 53, 59, 75 and 98. *Exposure:* A narrow band of low frequency (300–600 cps) continuous noise at 105 db for 48 hours. *Results:* Moderate external hair and supporting cell injury is present for two specimens in the lower part of the second turn with only mild changes in nearby areas. One specimen is practically within normal limits while the remainder show slight injuries in the lower and middle parts of the second turn.

Appendix B *Pre-exposure thresholds in SPL and final threshold shifts (PTS)*

Exposure	Days after noise	Frequency in kc/sec									
		0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	$\overline{PTS}$
¼ hr	P	44.8	19.8	4.0	−8.5	−14.0	−19.2	−25.8[a]	20.5	—	
b b n	46	−3.8	−5.0	3.0	8.8	−5.0	3.8	−1.2	−2.5	—	−0.2
c	P	44.8	19.8	25.8	−6.0	−20.2	13.0	−27.0[a]	16.8	—	
115 db	152	1.9	3.8	−13.1	21.9	28.1	28.8	10.0	5.6	—	10.9
	P	44.8	19.8	12.0	−8.5	−19.0	13.0	−23.2[a]	19.8	—	
	149	−4.4	5.0	−1.9	26.9	18.1	0.0	1.9	7.5	—	6.2
½ hr	P	44.8	21.0	−0.5	−16.0	−24.0	19.2	−24.5[a]	−5.5	—	
b b n	118	0.0	−2.5	4.4	21.2	47.5	45.0	12.5	11.2	—	14.6
c	P	34.8	13.5	−0.5	−16.0	−15.2	−15.5	28.8[a]	−5.5	—	
115 db	118	6.9	5.0	3.1	11.9	40.6	43.8	27.5	−5.6	—	16.6
	P	48.5	19.8	−1.8	−12.2	−27.8	18.0	−22.0[a]	−13.0	—	
	75	−8.1	−1.9	5.0	6.2	18.8	3.8	0.0	0.0	—	3.0
	P	31.6	19.4	7.4	−9.1	−23.6	−7.8	14.6	2.0	38.0	
	84	−7.0	−8.7	1.3	0.8	3.4	−3.0	0.8	5.5	−13.5	2.7
	P	25.6	20.4	−4.6	−10.1	−16.6	15.8	−15.6	6.0	15.0	
	84	4.0	5.0	16.0	20.0	29.0	−2.0	1.0	2.0	6.0	9.1
	P	22.6	23.4	9.4	−11.1	−13.6	−13.8	12.6	5.0	12.0	
	84	3.0	−8.0	2.0	21.0	12.0	35.0	−2.0	15.0	70.0	9.8
2 hr	P	41.0	14.8	7.0	0.2	19.0	15.5	27.0[a]	−23.0	—	
b b n	118	7.5	10.0	13.1	34.4	46.4	44.4	20.6	0.0	—	22.0
c	P	44.8	19.8	4.5	−8.5	12.5	16.5	18.2[a]	9.2	—	
115 db	145	0.0	3.8	5.0	26.9	48.5	43.5	8.8	−5.6	—	15.4
	P	44.8	19.8	4.0	−13.5	−14.0	−10.5	−18.4[a]	−11.8	—	
	171	8.1	21.9	23.0	48.1	50.6	48.8	49.4	43.8	—	36.7
	P	41.8	21.0	3.2	−6.0	19.0	19.2	23.2[a]	14.2	—	
	117	11.2	19.4	53.8	48.8	53.8	53.1	50.6	46.2	—	42.1
	P	31.6	16.4	1.4	9.1	15.6	7.8	8.6	3.0	11.0	
	84	11.8	15.0	35.0	58.8	63.2	51.0	50.0	50.0	68.7[b]	[illegible]
	P	32.6	13.9	3.9	12.1	14.6	17.3	17.1	6.5	12.5	
	84	17.0	19.5	22.5	55.0	62.1	54.3	43.5	50.0	67.0[b]	40.5
	P	39.1	16.9	3.4	−12.1	17.1	−19.3	−17.1	−0.5	11.5	
	84	13.9	25.5	31.0	62.2	62.5	54.5	56.7	55.0	[illegible][b]	[illegible]
	P	29.6	10.4	1.4	12.1	−19.6	−13.8	27.6	−12.0	1.0	
	84	10.0	21.0	29.0	65.2	60.0	67.0	68.2	56.0	76.5[b]	[illegible]
	P	34.6	21.4	7.4	2.1	3.6	8.2	−3.6	15.0	18.0	
	84	35.0	41.0	56.0	51.6	40.6	25.6	40.4	−9.2	[illegible]	[illegible]
	P	31.6	16.9	7.9	8.6	7.6	13.8	−13.6	2.5	18.5	
	84	20.1	28.7	61.0	66.0	[illegible]	[illegible]	[illegible]	35.0	12.5	[illegible]

Appendix B (cont.)

Cat no	Exposure	Days after noise	Frequency in kc/sec									
			0.125	0.25	0.5	1.0	2.0	4.0	8.0	16.0	32.0	$\overline{PTS}$
78		P	23.1	16.9	6.9	−6.6	−12.6	−17.3	−16.6	−4.5	15.0	
		84	7.5	4.5	6.5	11.5	46.0	53.5	12.0	3.5	10.0	18.1
80		P	34.6	22.4	−0.6	−2.1	3.6	−20.8	−15.6	3.0	36.0	
		84	8.0	15.0	51.4	37.0	35.0	17.0	31.0	6.0	5.0	28.8
86	8 hr	P	27.6	18.4	9.4	−1.1	−14.6	0.2	−17.6	−2.0	11.0	
	b b n	84	19.0	17.0	21.0	63.1	69.1	48.1	62.1	58.5	68.5[b]	45.1
90	c	P	26.6	17.4	7.4	−9.1	−15.6	−10.8	−17.6	−6.0	10.0	
	115 db	84	18.0	28.0	47.0	82.4	77.5	75.5	31.6	80.5	63.5[b]	62.9
92		P	23.6	11.4	−1.6	−14.1	−19.6	−17.8	−15.6	−5.0	10.0	
		84	33.1	33.0	57.1	67.0	66.0	66.1	55.2	52.5	57.5[a]	53.8
102		P	28.6	18.4	9.4	8.1	−18.6	−2.8	16.6	1.0	17.0	
		84	8.0	17.0	31.0	19.0	47.0	35.0	36.0	26.0	5.0	31.1
107		P	30.6	16.1	3.4	−11.1	20.6	−9.8	−4.6	−1.0	18.0	
		84	19.0	15.0	29.0	60.0	56.0	16.0	25.0	6.0	11.0	32.0
112		P	28.6	15.4	9.4	4.1	10.6	10.8	10.6	−11.0	15.0	
		84	9.0	9.0	5.0	18.0	50.0	13.0	5.0	11.0	4.0	18.8
39	2 hr	P	30.6	18.4	8.4	6.1	15.6	6.8	18.6	4.0	9.0	
	b b n	84	15.0	22.0	43.0	44.0	39.0	40.0	29.0	6.0	10.0	28.2
50	S	P	31.6	23.4	6.4	9.1	12.6	17.8	18.6	6.0	9.0	
	16 doses	84	4.0	5.0	4.0	9.0	11.0	22.0	4.0	1.0	3.0	6.0
69	7½ min	P	28.6	14.4	3.4	3.1	15.6	15.8	6.6	3.0	12.0	
	1 hr IEI	84	1.0	9.0	4.0	14.0	35.0	48.0	12.0	10.0	4.0	16.0
72	115 db	P	29.6	15.4	2.4	−13.6	27.6	12.8	21.6	9.0	8.0	
		84	9.0	1.0	2.0	1.0	5.0	3.0	1.0	8.0	0.0	2.8
52	2 hr	P	26.6	10.4	0.4	7.1	16.6	11.8	18.6	7.0	10.0	
	b b n	84	2.0	4.0	0.0	9.0	0.0	6.0	1.0	2.0	4.0	0.0
56	S	P	31.6	20.4	5.4	12.1	9.6	4.8	15.6	5.0	13.0	
	16 doses	84	2.0	9.0	16.0	25.0	7.0	0.0	3.0	6.0	1.0	5.8
62	7½ min	P	27.6	14.4	8.4	8.1	11.6	1.8	−13.6	1.0	17.0	
	6 hr IEI	84	4.0	6.0	17.0	38.0	41.0	16.0	1.0	6.0	5.0	11.8
74	115 db	P	43.6	22.4	5.4	10.1	16.6	15.8	19.6	8.0	12.0	
		84	1.0	0.0	4.0	13.0	13.0	34.0	1.0	1.0	1.0	8.4
60[c]	2 hr	P	32.6	13.9	3.9	12.1	14.8	17.3	17.1	6.5	12.5	
	b b n	109	5.0	0.5	6.5	0.0	1.4	6.5	0.5	0.5	3.5	0.8
63[c]	S	P	39.1	16.3	3.4	12.1	17.1	13.3	17.1	0.5	11.5	
	16 doses	109	0.5	1.5	3.0	0.0	1.5	10.5	0.5	3.5	1.5	1.8
76[c]	7½ min	P	[illegible]	16.9	7.9	8.6	7.6	13.8	13.6	2.5	18.5	
	24 hr IEI	109	[illegible]	3.5	6.5	2.5	4.0	8.0	12.0	2.5	4.5	3.5

ndix B (cont)

no	Exposure	Days after noise	Frequency in kc/sec 0 125	0 25	0 5	1 0	2 0	4 0	8 0	16 0	32 0
8[c*]	115 db	P	23 1	16 9	6 9	-6 6	-12 6	17 3	-16 6	-4 5	15 0
		109	6 5	3 5	1 5	0 5	-4 0	-1 5	4 0	-1 5	0 0
1	8 hr	P	25 6	10 4	-2 6	-10 1	-18 6	-29 8	-15 6	12 0	11 0
	n b n	84	-2 0	-2 0	5 0	2 0	-1 0	12 0	7 0	7 0	7 0
0	c	P	24 6	14 4	6 4	-3 1	-5 6	-13 8	-16 6	-3 0	17 0
	105 db	84	2 0	3 0	4 0	1 0	10 0	2 0	0 0	2 0	9 0
9		P	26 6	17 4	10 4	-5 1	-18 6	-17 8	19 6	-3 0	20 0
		84	-2 0	-1 0	-3 0	-5 0	6 0	17 0	5 0	1 0	1 0
9		P	32 6	13 4	0 4	0 9	-15 6	9 8	22 6	-7 0	11 0
		84	-2 0	3 0	4 0	-3 0	-1 0	-10 0	4 0	-2 0	8 0
3	48 hr	P	22 6	6 4	-1 6	-7 1	22 6	-21 5	21 6	-13 0	8 0
	n b n	84	2 0	3 0	2 0	8 0	-3 0	-3 0	5 0	8 0	12 0
9	c	P	24 6	9 4	1 4	-4 1	-16 6	22 8	-21 6	-6 0	11 0
	105 db	84	-1 0	-1 0	-1 0	-10 0	-2 0	6 0	2 0	-10 0	2 0
5		P	29 6	14 4	6 4	-11 1	-15 6	-17 8	19 6	-10 0	11 0
		84	5 0	3 0	6 0	4 0	5 0	12 0	4 0	8 0	15 0
8		P	29 6	13 4	2 6	-8 1	16 6	15 8	-19 6	-6 0	11 0
		84	4 0	4 0	9 0	4 0	1 0	-4 0	-2 0	1 0	12 0

In these instances frequency was 8 8, not 8 0 kc/sec P pre exposure

No response at audiometer s maximum

Later used in 2 hr cont exposure

★

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 177

FOKUS-TESTE DER TONSILLEN BEI FOKAL-INFEKTION

VON

Y. NOSAKA

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 177

FOKUS-TESTE DER TONSILLEN BEI FOKAL-INFEKTION

BY

YASUTSUGU NOSAKA

Aus der Universitatsklinik fur Hals Nasen und Ohrenkranke
(Direktor Prof Dr Y Nosaka) Kumamoto Japan

Printed in Japan
BY SHUHANSHYA
KUMAMOTO *1963*

INHALT

EINLEITUNG

Der Begriff der Fokal-Infektion, dass allgemeine Erkrankungen oft durch lokale Infektion hervorgerufen worden sind, ist schon von altersher bekannt, und zwar seit dem Zeitalter, wo noch die medizinischen Kenntnisse äusserst dürftig waren. Sekundäre Erkrankungen, die als Folge-Krankheit der Herd-Infektion annehmbar sind, sind heute zahlreich und als wichtige Probleme viel fach in der Klinik diskutiert. Die ätio-pathologische Auffassung, die den Primärherd zwangsläufig mit den daraus entwickelten, sekundären Erkrankungen zu verbinden versucht, ist auf Umwegen jetzt zum sensus communis der Klinik geworden. Die Auffassung ist auch dadurch verständlich, dass die dem Primärherd hinzutretenden sekundären Erkrankungen bei seiner Verschlimmerung bzw. Wiederaufflackerung exacerbieren, und umgekehrt, durch Entfernung des Primärherdes zur Besserung bzw. Heilung kommen.

Die Auffassung der Herd-Infektion schliesst jedoch insofern in sich noch einen schwachen Punkt ein, da die experimentelle Grundlage für den Entstehungsmechanismus noch gar nicht erhärtet worden ist. Nicht allein dadurch, sondern wegen des Fehlens der exakten diagnostischen Methode musste man sich bisher nur auf Diagnosis ex juvantibus halten. Nach der Einführung der Allergie-Theorie, der Kenntnis über Reilly-Phänomen und der Stress-Theorie haben in den letzten Jahren die Forschungen über die experimentelle Herd-Infektion rasche Fortschritte gemacht, wodurch auch die Pathogenese allmählich aufgeklärt werden konnte.

Als Fokus der Herd-Infektion kommen nun die chronische Tonsillitis, Zahnerkrankung oder Nebennasenhöhlenentzündung, d. h. sogenannte Kopfherde, in Frage. Es ist jedoch ganz klar, dass vor allem chronische Tonsillitis den Kern der Fokus darstellt. Es ist aber nicht immer der Fall, dass solche Herde stets für eine Fokal-Infektion verantwortlich sein dürfen.

Für die Aufstellung der therapeutischen Richtlinien kommt es aber sehr darauf an, beim Vorhandensein der chronischen Tonsillitis und damit zusammen auch der auf Fokal-Infektion verdächtigen allgemeinen Erkrankungen a priori die Herd-Pathogenität der Tonsillen klar zu machen. Trotzdem ist die Diagnose nicht immer so leicht. Selbst bei Diagnosis ex juvantibus, die auf Grund des Heilzustandes von sekundären Erkrankungen durch Tonsillektomie nach der erfahrungsgemaess als sehr zuverlässig ist, ist denn noch die Diagnose-Stellung äusserst schwer, besonders schwer in solchem Fall, bei dem der Primärherd entweder schon über das Operationsfeld hinausragte oder den sekundären Fokus bildete, aber auch sekundäre Krankheiten bereits zur veralteten, irreversiblen Krankheitsveränderung gekommen sind.

Zur Beurteilung, ob fragliche Tonsillen tatsächlich den Fokus darstellen, hat man früher das Augenmerk vielfach auf den lokalen pathologischen Befund gerichtet, der aber bloss ein Befund der chronischen Tonsillitis, nicht aber für Tonsillen als Fokus charakteristisch ist. Deshalb hat man später verschiedene Teste vorgenommen. Es liess sich jedoch noch nicht sagen, dass die allgemeine Reaktion direkt mit dem Krankheitsherd der Tonsillen zu tun hat.

Ausserdem noch hat sich der Einfluss von sekundaren Krankheiten daran nicht unwesentlich beteiligt

Neuerdings hat man sich vielfach bestrebt durch Provokation des verdachtigen Primarherdes oder umgekehrt durch Blockierung bzw Annulation der Einflusse die dem Primarherd entstammen die Befund des Primarherdes oder die Symptome der sekundaren Erkrankungen dadurch zu beeinflussen Nicht nur dies sondern es wurden auch die Veranderungen von Korpertemperatur Blut, Urin Elektrokardiogramm usw verfolgt

So sind heute die Provokations bzw Annulationsdiagnostik aber auch die kombinierte Diagnostik dieser beiden Methoden in steigendem Ausmass erprobt

Denn das Prinzip der Methoden ist kurz gesagt Durch Reize verschiedener Art auf den Fokus werden die Bakterien bzw toxische Produkte aber auch destruktive Produkte der Lymphozyten oder der antigenaktive Fokus Inhalt in die Korperflussigkeit getrieben Andererseits wird dadurch die Dystonie des autonomen Nervensystems hervorgerufen Die dabei entstandene Veranderung der allgemein als Abwehrreaktion angenommenen Vitalreaktion und die Veranderung der Symptome von sekundaren Erkrankungen werden beobachtet Des weiteren werden die von dem Fokus ausgelosten Impulse blockiert um damit den Einfluss auf Fernsymptome zu beobachten und die Pathogenitat des Herdes festzustellen Bezuglich dieser diagnostischen Prufungsmethoden hat GUTZEIT die Pathologie der Herd Infektion zu einer Noxen Theorie zusammengefasst was uns gerade die theoretische Grundlage schuf

Fur den Fokus Test gibt es verschiedene Klassifikationen z B nach subjektiven und objektiven Symptomen oder nach der Pathogenitat aber auch nach der allgemeinen Reaktion sowie der lokalen Herdreaktion Als Methodik werden auch physikalische medikament se oder immunserologische Methode usw angewandt. Im folgenden werden wir aber unsere Untersuchungsergebnisse hauptsachlich nach meiner eigenen Klassifikation mitteilen

Die von uns behandelten sekundaren Erkrankungen bei Herd Infektion sind die Krankheiten bei denen die Ansichten der Autoren weitgehend einig sind und zwar als rheumatische Krankheiten die rheumatische Arthritis Sehnenentzundung insbesondere die Achillessehnenentzundung oder Myalgie und als Herzkrankheiten die Endokarditis Mitralinsuffizienz oder Arhythmie und als Nierenkrankheiten die Glomerulonephritis oder zirkumskripte Nephritis und noch als Hautkrankheiten Erythema nodosum sowie Urticaria und als Augenkrankheiten die Iritis sonst chronische Subfebrilitaten

UNTERSUCHUNGSERGEBNISSE

I Allgemeine Diagnostik

1 *Anamnese* Falls in der früheren Anamnese irgend ein Zusammenhang zwischen Fokus und Sekundärleiden bestünde so wurde die Vertrauenswürdigkeit dieser Beziehung weiterhin nicht nur durch Diagnosis ex juvantibus nach der Tonsillektomie sondern auch auf Grund der Untersuchungsergebnisse eingehend geprüft Auf diese Weise konnte die Korrelation bei Fällen der Tonsillitis chronica focalis die anamnestisch schon die Beziehung vermuten liess in 59 8 % und bei Fällen auffallenden tonsilogenen Ursprungs sogar in 77 9 % jedoch bei verdächtigen Fällen nur in 22 1 % festgestellt werden Es hat sich also herausgestellt, dass die Anamnese wie schon PARADE darauf hinwies eine grosse diagnostische Bedeutung hat. Nach PARADE soll die Diagnose nämlich bei der genauen Anamnese in 3/4 der ganzen Fälle sicher gestellt werden Deshalb darf die Herd Infektion selbst bei solchen Fällen in denen der Zusammenhang noch nicht klar ist nicht einfach abgelehnt werden

2 *Blutbefund* Die Zunahme der Leukozytenzahl um 1000 und noch mehr wurde bei Tonsillitis chronica simplex in 16 7 % und bei Tonsillitis chronica focalis in 24 % nachgewiesen Bei der Klassifikation der Leukozytenarten waren die Fälle der Leukozytose etwas mehr Die Plasmazellen die bei Tonsillitis chronica simplex noch niemals zu beobachten waren nahmen auch zu

DYACHENKO hat noch darauf hingewiesen dass bei Tonsillitis chronica insbesondere bei Herdreaktion eine regionäre Leukozytose an dem der Mandel ganz nahe liegenden Seitenhals nachweisbar ist, während diese Erscheinung aber beim Fehlen der Mandel Entzündung oder nach der Tonsillektomie nicht mehr vorkommt so dass diese Erscheinung auch von der grossen diagnostischen Bedeutung ist. Da die periphere Leukozytenzahl in Fingerbeeren und Ohrlappen fast keinen Unterschied der Zahl aufwies so haben wir lokale Leukozytenzahl im Seitenhals und Ohrlappen miteinander verglichen Dabei zeigte es sich dass in positiven Fällen bei denen die Differenz an beiden Körperstellen mehr als 1000 war bei Tonsillitis chronica simplex in 11 8 % jedoch bei Tonsillitis chronica focalis in 28 6 % zu beobachten waren

Wenn die positiven Fälle aber nach 3 Stunden der Provokation weiterhin durch Ultrakurzwellen indirekt 10 Minuten lang bestrahlt wurden und dadurch die Leukozytenzahl nochmals um mehr als 1000 zunahm so wurden die Fälle nunmehr als positiv bezeichnet. Danach wurden positive Fälle bei Tonsillitis chronica simplex nur in 10 % dagegen bei Tonsillitis chronica focalis um das 5 Fache so häufig also in 50 % festgestellt. Bei dieser Provokation war auch die Erfolgsziffer der Tonsillektomie bei positiven Fällen der sekundären Erkrankungen sehr hoch wie 90 9 % Daher kommt dieser Art der Provokation eine grosse diagnostische Bedeutung zu nicht nur dies sondern die Methode ist auch als Indikator für Tonsillektomie zu verwerten

Ausserdem noch hat sich der Einfluss von sekundären Krankheiten daran nicht unwesentlich beteiligt

Neuerdings hat man sich vielfach bestrebt durch Provokation des verdächtigen Primärherdes oder umgekehrt durch Blockierung bzw Annulation der Einflusse die dem Primärherd entstammen die Befund des Primärherdes oder die Symptome der sekundären Erkrankungen dadurch zu beeinflussen Nicht nur dies sondern es wurden auch die Veränderungen von Körpertemperatur Blut Urin Elektrokardiogramm usw verfolgt

So sind heute die Provokations- bzw Annulationsdiagnostik aber auch die kombinierte Diagnostik dieser beiden Methoden in steigendem Ausmass erprobt

Denn das Prinzip der Methoden ist kurz gesagt Durch Reize verschiedener Art auf den Fokus werden die Bakterien bzw toxische Produkte aber auch destruktive Produkte der Lymphozyten oder der antigenaktive Fokus Inhalt in die Korperflussigkeit getrieben Andererseits wird dadurch die Dystonie des autonomen Nervensystems hervorgerufen Die dabei entstandene Veränderung der allgemein als Abwehrreaktion angenommenen Vitalreaktion und die Veränderung der Symptome von sekundären Erkrankungen werden beobachtet Des weiteren werden die von dem Fokus ausgel sten Impulse blockiert um damit den Einfluss auf Fernsymptome zu beobachten und die Pathogenität des Herdes festzustellen Bezuglich dieser diagnostischen Prufungsmethoden hat GUTZEIT die Pathologie der Herd Infektion zu einer Noxen-Theorie zusammengefasst was uns gerade die theoretische Grundlage schuf

Fur den Fokus Test gibt es verschiedene Klassifikationen z B nach subjektiven und objektiven Symptomen oder nach der Pathogenität aber auch nach der allgemeinen Reaktion sowie der lokalen Herdreaktion Als Methodik werden auch physikalische medikament se oder immunserologische Methode usw angewandt Im folgenden werden wir aber unsere Untersuchungsergebnisse hauptsächlich nach meiner eigenen Klassifikation mitteilen

Die von uns behandelten sekundären Erkrankungen bei Herd Infektion sind die Krankheiten bei denen die Ansichten der Autoren weitgehend einig sind und zwar als rheumatische Krankheiten die rheumatische Arthritis Sehnenentzündung insbesondere die Achillessehnenentzündung oder Myalgie und als Herzkrankheiten die Endokarditis Mitralinsuffizienz oder Arhythmie und als Nierenkrankheiten die Glomerulonephritis oder zirkumskripte Nephritis und noch als Hautkrankheiten Erythema nodosum sowie Urticaria und als Augenkrankheiten die Iritis sonst chronische Subfebrilitäten

UNTERSUCHUNGSERGEBNISSE

I Allgemeine Diagnostik

1 *Anamnese* Falls in der früheren Anamnese irgend ein Zusammenhang zwischen Fokus und Sekundärleiden bestünde so wurde die Vertrauenswürdigkeit dieser Beziehung weiterhin nicht nur durch Diagnosis ex juvantibus nach der Tonsillektomie sondern auch auf Grund der Untersuchungsergebnisse eingehend geprüft Auf diese Weise konnte die Korrelation bei Fällen der Tonsillitis chronica focalis die anamnestisch schon die Beziehung vermuten liess in 59 8 % und bei Fällen auffallenden tonsilogenen Ursprungs sogar in 77 9 % jedoch bei verdächtigen Fällen nur in 22 1 % festgestellt werden Es hat sich also herausgestellt, dass die Anamnese wie schon PARADE darauf hinwies eine grosse diagnostische Bedeutung hat Nach PARADE soll die Diagnose nämlich bei der genauen Anamnese in 3/4 der ganzen Fälle sicher gestellt werden Deshalb darf die Herd Infektion selbst bei solchen Fällen in denen der Zusammenhang noch nicht klar ist, nicht einfach abgelehnt werden

2 *Blutbefund* Die Zunahme der Leukozytenzahl um 1000 und noch mehr wurde bei Tonsillitis chronica simplex in 16 7 % und bei Tonsillitis chronica focalis in 24 % nachgewiesen Bei der Klassifikation der Leukozytenarten waren die Fälle der Leukozytose etwas mehr Die Plasmazellen die bei Tonsillitis chronica simplex noch niemals zu beobachten waren nahmen auch zu

DYACHENKO hat noch darauf hingewiesen dass bei Tonsillitis chronica insbesondere bei Herdreaktion eine regionäre Leukozytose an dem der Mandel ganz nahe liegenden Seitenhals nachweisbar ist, während diese Erscheinung aber beim Fehlen der Mandel Entzündung oder nach der Tonsillektomie nicht mehr vorkommt so dass diese Erscheinung auch von der grossen diagnostischen Bedeutung ist Da die periphere Leukozytenzahl in Fingerbeeren und Ohrlappen fast keinen Unterschied der Zahl aufwies so haben wir lokale Leukozytenzahl im Seitenhals und Ohrlappen miteinander verglichen Dabei zeigte es sich dass in positiven Fällen bei denen die Differenz an beiden Körperstellen mehr als 1000 war bei Tonsillitis chronica simplex in 11 8 % jedoch bei Tonsillitis chronica focalis in 28 6 % zu beobachten waren

Wenn die positiven Fälle aber nach 3 Stunden der Provokation weiterhin durch Ultrakurzwellen indirekt 10 Minuten lang bestrahlt wurden und dadurch die Leukozytenzahl nochmals um mehr als 1000 zunahm so wurden die Fälle nunmehr als positiv bezeichnet. Danach wurden positive Fälle bei Tonsillitis chronica simplex nur in 10 % dagegen bei Tonsillitis chronica focalis um das 5 Fache so häufig also in 50 % festgestellt Bei dieser Provokation war auch die Erfolgsziffer der Tonsillektomie bei positiven Fällen der sekundären Erkrankungen sehr hoch wie 90 9 % Daher kommt dieser Art der Provokation eine grosse diagnostische Bedeutung zu nicht nur dies sondern die Methode ist auch als Indikator für Tonsillektomie zu verwerten

Die Blutkörperchen Senkungsgeschwindigkeit (BSG) war bei Tonsillitis chronica simplex nur in 18 4 % dagegen bei Tonsillitis chronica focalis in 56 9 % beschleunigt besonders deutlich bei Nephritis und Rheumatismus Ferner wurden die BSG bie den Falle der Herd Infektion durch Tonsillektomie um das 3 Fache und noch mehr gebessert was aber damit allein nicht als ein diagno stische Charakteristikum anzunehmen ist

3 *Elektrokardiogram* (EKG) Bei 36 Fallen der Tonsillitis chronica simplex und bei 109 Fallen der von sekundaren Krankheiten begleitenden chro nischen Tonsillitis wurde der Arbeit Belastungsversuch nach SCHLOMKA vor genommen und es konnte dadurch in 30 3% der Falle ein abnormaler Befund und zwar in 17 Fallen die Veranderung von ST am Haufigsten dann in 5 Fallen die Veranderung von P haufig und noch in je 2 Fallen die Verflachung von T die Verlangerung von QRS die Niederspannung sowie die ventrikularen Extra systolen usw festgestellt werden Solche Anomalien der EKG traten aber bei Tonsillitis chronica simplex nur in 5 6 % dagegen bei Tonsillitis chronica focalis in 43 1 % auf und dann in der Reihenfolge der Herzkrankheiten Sub febrilitaten Nephritis und Rheumatismus immer haufiger auf Nach der Ton sillektomie wurden die EKG Anomalien in 55% gebessert besonders aber wur den die Anomalien bei Nephritis und Subfebrilitaten g instig beeinflusst jedoch mit Ausnahme von 2 Fallen der Tonsillitis chronica simplex Die Besserung wurde besonders haufig bei der Verflachung von T und der Niederspannung aber auch bei der Veraenderung von ST beobachtet

Bei 25 Fallen der Tonsillitis chronica focalis wurde noch die Provokation der Mandel vorgenommen

Dadurch wurde die Veranderung durch Provokation bei 3 Fallen durch Ultra kurzwellen und bei 2 Fallen durch Hyaluronidase Massage auftraten also im ganzen in 20 % beobachtet besonders deutlich aber bei Herzkrankheiten wobei die Veranderung von QRS und die Verflachung von T also uberhaupt die Veranderung des Ventrikel Komplex festgestellt wurde Dabei wurde die Veranderung am fruhesten schon in 3 Stunden nach der Provokation und bei sonstigen Fallen auch nach 3 Tagen durch Registrierung festgestellt (Tabelle 1)

Von diesen Fallen wird das EKG von Fall 3 in Abb 1 gezeigt

Ganz umgekehrt dazu wurde die deutliche Veranderung von EKG nach der Lacunenspulung in einem Fall von 3 Herzkranken und die wahrscheinlich ge besserte Veranderung in einem anderen Fall beobachtet Da solche temporare Verschlimmerung bzw Besserung des Ventrikel Komplexes durch Provokation bzw Annulation schon in kurzer Zeit nach dem Eingriff auftrat so ist die Annahme berechtigt dass die Veranderung wahrscheinlich auf die durch auto nomen Nervenfaktor bedingte Abnahme der Blutmenge im Koronalkreislauf und damit auf die durch O_2 Mangel im Herzmuskel hervorgerufene Stoffwech selstorung zuruckzufuhren ist und dass die Veranderung der Vorhofspulswellen aber auf der durch ahnlichen Mechanismus bedingten funktionellen Dilatation des Vorhofs beruhen darf

Die Erfolgsziffer der abnormalen Falle nach der Tonsillektomie bei sekun daren Krankheiten betrug 91 7 % bei Rheumatismus und Subfebrilitat sogar 100 % Beim Herzleiden konnte auch in 3 von 4 Fallen der Erfolg beobachtet werden Da der Besserungserfolg bei den Fallen die noch keine Anomalien

TABELLE 1 *Veranderung von Elektrokardiogram bei der Provokation*

	Krankheiten	Lj	Geschl	Provokationsmethode	vor Provokation	nach Porvokation	EKG-Veranderung nach der Tonsillektomie	Erfolg der Tonsillektomie bei sekundären Krankheiten
Tonsillogene Erkrankungen	Rheuma	29	♀	H M	$P_{II,III}$ positiv	3 Tage nach der Provokation $T_{II,III}$ herabgesetzt $P_{II,III}$ positiv	nach 7 Tagen T erhöht u $P_{II,III}$ positiv	günstig
		21	♀	UKW	kein abnormaler Befund	nach 3 Tagen Potentialdiff v QRS I II, III erniedrigt	unklar	günstig
	Nephritis	25	♂	H M	kein abnormaler Befund	nach 5', 15', 30' u 1^h noch keine Veranderung, jedoch nach 3^h Verflachung von $T_{I\ III}$, V_4 V_6, bes bei Belastungs EKG deutlich, nach 2 Tage keine Anomalie	keine Anomalie	günstig
	Herzleiden	15	♂	UKW	Hypertrophie d linken Herzens	nach 3 Tagen $P_{II,III}$ hoch Pulmonal-P T_{III} auch hoch	unklar	unklar
		17	♂	UKW	QT verlängert u $T_{V_1-V_3}$ verflacht	nach 3^h Umkehrung von T_{V_1}, V_2, V_3	keine Anomalie	günstig
Tonsillitis chronica simplex		23	♀	UKW	keine Anomalie	nach 3^h $P_{II,III}$ V_4 V_5 spitzig u hoch, bes deutlich bei Belastungs EKG	$P_{II,III}$ allmahlich niedriger	

H M Hyaluronidase-Massage UKW Ultrakurzwellen

zeigten auch sehr hoch war, so konnte die Herd Infektion nicht negiert werden Es ist deshalb anzunehmen, dass die Anomalien von EKG als Herd-Diagnostik bedeutungsvoll und die durch Provokation bzw Annulation herbeigefuhrten Veranderungen noch mehr sinnvoll sind

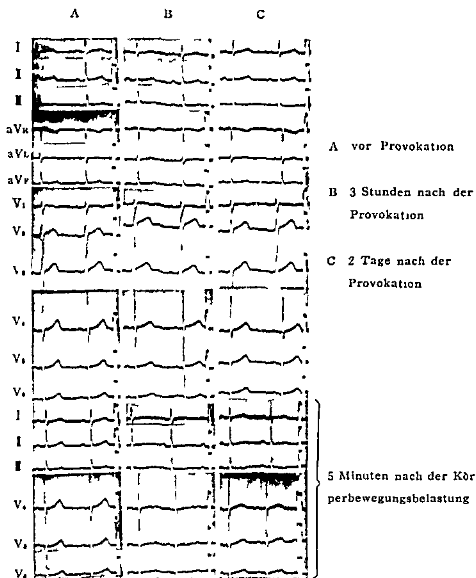


ABB 1 EKG vor und nach der Provokation (Fall 3 25 J ♂ Tonsillogene Nephritis)

1 *Funktionsprüfung des autonomen Nervensystems*

Die Ansichten über den Entstehungsmechanismus der Herd Infektion sind heute noch geteilt Die Beteilung des autonomen Nervensystems daran ist—sei es die Theorie der Relationspathologie sei es die Lehre der Neurodystrophie oder aber auch die Reilly s Phänomen oder Stress Theorie—sicher gross, so dass die Art und Weise der autonomen Nervenfunktion bei diesen Erkrankungen von grossem Interesse sind

Zur Funktionsprüfung sind die Resultate der pharmakodynamischen Unter

suchung besonders verwertbar So zeigten 10 Fälle der Tonsillitis chronica simplex sämtlich die Amphotonie und 20 Fälle der Tonsillitis chronica focalis auch in 55 % einen gleichnamigen Typus und sonstige Fälle in 25 % die para sympathische Unruhe, sonst noch amphotonische Unruhe oder Parasympathi kotonie, also überhaupt die unregelmässige Steuerung der autonomen Nerven funktion, was darauf hinzudeuten ist, dass bei der Entstehung der Herd In fektion die autonome Nervenfunktion eine grosse Rolle spielt Bei der physi kalischen Prüfung war aber die Abweichung von der Tonsillitis chronica sim plex nicht deutlich feststellbar

5 *Thorn Test* Die durch Herd Infektion herbeigeführten sekundären Krankheiten umfassen manche Dysadaptations- und Kollagenkrankheiten Deshalb hat man behauptet, dass der Thorn Test dabei stark positiv ausfallen muss Der Test fiel bei der chronischen Tonsillitis, die vom Sekundärleiden begleitet worden ist, in 54 2 %, und sogar bei Herdkrankheiten tonsillogenen Ursprungs sogar in 75 % positiv aus, also viel häufiger als bei Tonsillitis chronica simplex oder einfacher Tonsillen Hypertrophie Der Test wurde aber nach der Tonsillektomie in der Überzahl der Fälle negativ, so dass man ver muten kann, dass bei der Herd-Infektion die Neigung der Beteiligung von Nebennierenrinden-Insuffizienz recht gross ist.

6 *Weltmann-Reaktion* Bei dieser Reaktion handelt es sich um eine Hitzekoagulationsreaktion, die sich lediglich auf Veränderung der Bestandteile von Serum Proteinen bezieht. Bei der Herd Infektion ist die Dysproteinämie sehr beachtlich, so dass die Ausführung der Reaktion ganz bedeutungsvoll ist. Die Reaktion fiel bei Tonsillitis chronica simplex in 26 1 %, jedoch bei tonsil logenen Herd-Infektionen in 54 3 % d h um das Doppelte so hoch, positiv aus, wobei in sämtlichen positiven Fällen nur eine Verlängerung des Koagula tionsbandes zu beobachten war Nach der durch Hyaluronidase-Massage ausgelösten Provokation wurde die Reaktion bei Tonsillitis chronica simplex sämtlich negativ, blieb dagegen bei tonsillogenen Herd Infektionen noch in 33 3 % positiv Diese Erscheinung war besonders signifikant bei Nephritis Mit Rücksicht auf eine hohe Erfolgsziffer nach der Tonsillektomie bei positiven Fällen wird grosses Gewicht auf die positive Reaktion nach der Provokation gelegt

7 *Analyse der Fraktionen von Plasmaproteinen* γ-Globuline haben eigentlich mit dem Antikörper zu tun Des weiteren ist die Analyse der Ei weiss-Fraktionen aus dem Standpunkt der Dysproteinämie interessant. Das Charakteristische der elektrophoretischen Analyse nach TISELIUS bei Tonsil litis chronica focalis liegt darin, dass gegenüber der Tonsillitis chronica sim plex Albumin—mit Ausnahme der Nephritis—abnimmt und γ-Globulin zunimmt.

Nach der Tonsillektomie zeigte Albumin die Abweichung etwas mehr nach der Obergrenze des Schwankungsumfangs bei Tonsillitis chronica simplex und γ-Globulin nahm dabei ab Die Neigung der Albumin abnahme und γ-Globulin zunahme war auch bei Herd Infektionen nach der an Mandel erfolgten Ultra kurzwellen Provokation wahrnehmbar Die elektrophoretische Analyse der Eiweiss Fraktionen durch Provokation ist deswegen diagnostisch mehr oder weniger wertvoll jedoch viel weniger bedeutungsvoll als des Index der Leukozyten, BSG und Körpertemperatur

8 *Bestimmung der Mukoproteine im Serum*. Mukoproteine bleiben bei

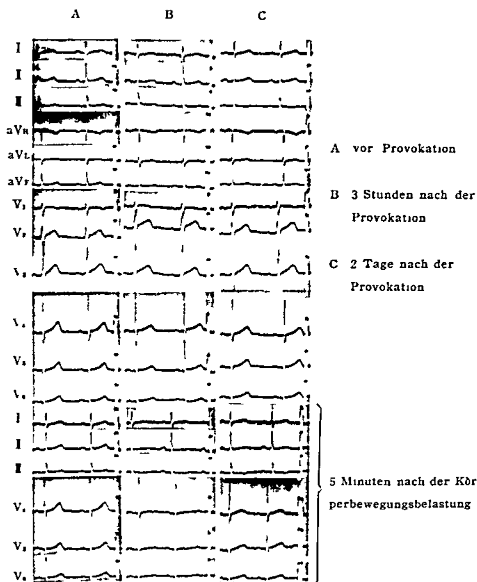


Abb 1 EKG vor und nach der Provokation (Fall 3 25 J ♂ Tonsillogene Nephritis)

4 *Funktionsprufung des autonomen Nervensystems*

Die Ansichten uber den Entstehungsmechanismus der Herd Infektion sind heute noch geteilt Die Beteilung des autonomen Nervensystems daran ist—sei es die Theorie der Relationspathologie sei es die Lehre der Neurodystrophie, oder aber auch die Reilly s Phänomen oder Stress Theorie—sicher gross, so dass die Art und Weise der autonomen Nervenfunktion bei diesen Erkrankungen von grossem Interesse sind

Zur Funktionsprufung sind die Resultate der pharmakodynamischen Unter

suchung besonders verwertbar So ze gten 10 Falle der Tonsil simplex samtlich die Amphoton e und 20 Falle der Tons llitis chr auch in 55 % einen gleichnamigen Typus u d son tige Falle in 2 sympathische Unruhe son t noch amphotonische Unruhe oder l kotome also uberhaupt die unregelmassige Steuerung der autonc funktion was darauf hinzudeuten ist, da s bei der Entstehung fektion die autonome Nervenfunktion eine grosse Rolle spielt. kalischen Prufung war aber die Abweichung von der Tonsillitis plex nicht deutlich feststellbar

5 *Thorn Test* Die durch He d Infektion herbeigef hr Krankheiten umfassen manche Dysadaptations und Kollag Deshalb hat man behauptet, dass der Thorn Test dabei stark muss Der Test fiel bei der chronischen Tonsillitis die vom begleitet worden ist, in 54 2 % und sogar bei Herdkrankheit Ursprungs sogar in 75 % positiv aus also viel haufiger als bei nica simplex oder einfacher Tonsillen Hypertrophie Der T nach der Tonsillektomie in der Uberzahl der Falle negat v muten kann dass bei der Herd Infektion die Neigung der Nebennierenrinden Insuffizienz recht gross ist

6 *Weltmann Reaktion* Bei dieser Reaktion handelt Hitzekoagulationsreaktion die sich lediglich auf Veranderun von Serum Proteinen bezieht. Bei der Herd Infektion ist sehr beachtlich, so dass die Ausf ihrung der Reaktion ganz Die Reaktion fiel bei Tonsillitis chronica simplex in 26 1 % logenen Herd Infektionen in 54 3 % d h um das Doppel aus wobei in samtlichen positiven Fallen nur eine Verlang tionsbandes zu beobachten war Nach der durch Hya ausgel sten Provokation wurde die Reaktion bei Tonsillit samtlich negativ blieb dagegen bei tonsillogenen Herd I 33 3 % positiv Diese Erscheinung war besonders signifi Mit Ri cksicht auf eine hohe Erfolgsziffer nach der Tonsille Fallen wird grosses Gewicht auf die positive Reakt on n gelegt.

7 *Analyse der Fraktionen von Plasmaproteinen* eigentlich mit dem Antik rper zu tun Des weiteren ist weiss Fraktionen aus dem Standpunkt der Dysproteinam Charakteristische der elektrophoretischen Analyse nach litis chronica focalis liegt darin dass gegen iber der T plex Albumin—mit Ausnahme der Nephritis—abnimmt un

Nach der Tonsillektomie zeigte Albumin die Abweic der Obergrenze des Schwankungsumfangs bei Tonsillitis γ-Globulin nahm dabei ab Die Neigung der Albumin a zunahme war auch bei Herd Infektionen nach der an kurzwellen Prov kation wahrnehmbar Die elektroph Eiweiss Fraktionen durch Provokation ist deswegen weniger wertvoll jedoch viel weniger bedeutungsvoll zyten BSG und K rpertemperatur

8 *Bestimmung der Mukoproteine im Serum* M

4 *Remky-Test* Die 1 Tropfen-Methode mit der Histaminlosung von 1 10,000 ist viel besser anwendbar als die Originalmethode Bei der Tonsillitis chronica simplex fiel der Test nur in 3 8 % positiv aus Bei der Verdunnung uber 1 20,000 war er nicht mehr positiv Bei der Fokal Infektion war der Test in 41 3 % und die Erfolgsziffer nach der Tonsillektomie in 89 5 % positiv Deshalb hat der Test gewissermassen diagnostischen Wert

III Topische Diagnose des Krankheitsherdes

1 *Befund der Tonsillen morphologische und elektronenmikroskopische Untersuchungen* Die Lacunen der Tonsillitis chronica focalis sind im Vergleich mit denen der Tonsillitis chronica simplex meist undeutlich und klein In manchen Fallen sind die Narbenbildung und der Druckschmerz in der Gegend von regionaren Lymphknoten und Jugularisgruben bemerkbar Jedoch ist noch kein Befund zu finden der zur Entscheidung der Herd-Affektionen ausschlaggebend ist. Dafuer ist aber nur die Eigenartigkeit der Mandelform massgebend So ist die Form der Mandel beim tonsillogenen Rheumatismus oder bei Herzleiden im Gegensatz zur Tonsillitis chronica simplex weniger kugelig und vielfach plattenformig oder hangeformig Der Vorsprungsgrad der Mandel nach dem Rachen ist aber nur klein Dagegen ist die kugelige Form bei Nephritis und Subfebrilitat in fast gleichem Ausmasse wie Tonsillitis chronica simplex haufig zu beobachten und der Vorsprungsgrad der Mandel bei Nephritis etwas geringer als bei denselben Fallen ist, jedoch unter allen Herd Infektionen am grossten Von den Fallen der Tonsillitis chronica focalis sind Volum und Gewicht bei Nephritis ganz gross bei Rheumatismus aber klein Die Mandel der subfebrilen Patienten und Herzkranken liegt in dieser Beziehung gerade mitten zwischen oben genannten Krankheiten Diese Neigung ist aber im allgemeinen etwas kleiner als bei Tonsillitis chronica simplex

Von der Bakterienflora in Lacunen sind die hamolytischen Streptokokken am haufigsten und zwar in 68 3 % gefunden Hamolytische Staphylokokken und Diplokokken sowie Leptothrix sind mehr als bei der Tonsillitis chronica simplex festgestellt worden Nach der Klassifikation der durch Tonsillenpunktion gewonnenen Zellen sind die Plasmazellen und Eosinophilzellen bei der Tonsillitis chronica focalis mehr als bei der Tonsillitis chronica simplex nachgewiesen Solche Zellen die mit der Antikorperbildung oder mit dem allergischen Geschehen in enger Beziehungen stehen bei der Fokal Infektion reichlich auftreten sind sie als diagnostisches Charakteristikum ganz wertvoll

Ferner wurden Epithelzellen der Lacunenschleimhaut und Parenchymzellen der Mandel elektronenmikroskopisch untersucht Hier werden aber nur einige Resultate mitgeteilt Bei Rheumatismus tonsillogenen Ursprungs bestehen die Schleimhaut Epithele aus 1—2 schichtigen Basalzellen Unter der Basalmembran laufen die Kollagenfibrillen durcheinander (Abb 4) Bei der Nephritis tonsillogenen Ursprungs treten aber interzellulare Lucken der Epithele auf Die Mikrozotten sind unregelformig und dick an deren Spitze die Partikelchen mit der hoher Elektronen Dichte sichtbar sind In Superficialzellen sind auch Vakuolen bemerkbar (Abb 5)

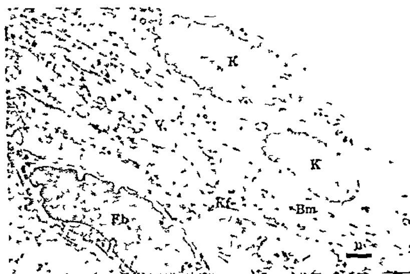


Abb 4 Epithelzellen der Schleimhaut
(Rheumatismus tonsillogenen Ursprungs)

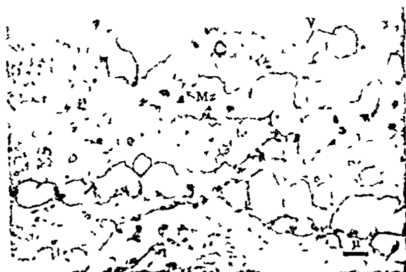


Abb 5 Schleimhautepithel der Lacunen
(tonsillogene Nephritis)

2 *Versuche mit Radioisotopen* Aus dem Grunde dass sich die Radioisotope in der Resorption, Aufspeicherung und der Elimination je nach den Krankheitszuständen etwas anders verhalten sind die Isotope gern zur Diagnose verschiedener Erkrankungen verwendet worden Die Forschungen über die Anwendung von Isotopen zur Fokus Diagnose sind aber nur wenig bekannt.

Wir haben nun ein festes Prüfungsmaterial, das aus dem 50 μC P^{32} haltigen anorganischen Phosphat, dessen Affinität relativ gross für das lymphatische Gewebe ist, und noch aus Milchzucker als Vehikel besteht, in die Lacuna su

perior bzw. Lacunen eingeschoben und damit die Radioaktivität der Mandel, des Blutes und des Urins bestimmt Die Resorptionsfähigkeit der Mandel zeigte dabei in 1—2 Stunden nach der Einfuhrung den maximalen Wert, so dass dieser Zeitraum nunmehr zur Grundlage der Bestimmung gewählt wurde Die Radioaktivität war aber bei normalen Tonsillen nur in 22 % der Fälle und bei Tonsillitis chronica simplex auch in 36 3 %, bei der Tonsillitis chronica focalis, besonders aber bei Nephritis, in 65 9 % nachweisbar Ferner war die Aktivität bei Subfebrilitäten in 40 6 % und bei Rheumatismus jedoch nur in 18 4 % feststellbar Die Radioaktivität des Blutes, die nach 3 Stunden ihren grossten Wert zeigte, war bei Tonsillitis chronica simplex etwas grosser als bei normalen Tonsillen Bei Tonsillitis chronica focalis, und zwar bei Rheumatismus, bei dem die Resorption aus der Mandel verzogert ist, und die Ausscheidung durch den Urin noch normal ist, zeigte die Radioaktivität nach 4 Stunden ihren maximalen Wert Dabei war jedoch C P M nicht so gross Bei Nephritis, bei der die Resorption noch gunstig, jedoch die Ausscheidung beeinträchtigt ist, war die Radioaktivität gross und dauerte sogar noch längere Zeit Die Radioaktivität des Urins erreichte, wie die des Blutes, gegenuber der subcutanen Injektion erst 1—2 Stunden später ihren maximalen Wert Bei der Nephritis war die Radioaktivität des Urins aber gering und es wurde sogar eine Verzogerung festgestellt Die Prufung uber den positiven Prozentsatz, indem 2 Kennzeichen, d h die Resorption aus Mandellacunen uber 30 % und in Blut uber 150 C P M positiv waren, ergab dass der totale Prozentsatz bei Tonsillitis chronica simplex nur 25 %, dagegen bei Tonsillitis chronica focalis, besonders aber bei Nephritis, 75 % und bei der Subfebrilität 62 5 % betrug, während er bei Rheumatismus nur 14 3 % zeigte, also niedrig war Da die Erfolgsziffer nach Tonsillektomie bei positiven Fällen sehr gross war 91 7 %, dagegen bei negativen Fällen viel kleiner war 41 7 %, so ist die Erfassung des Charakteristikum der sekundären Erkrankungen diagnostisch auch bedeutungsvoll

3 *Farbstoff-Entfarbungsreaktion in Tonsillen* Je 0 2 cc einer 0 8 %igen Methylenblaulosung oder einer 1 %igen 2—6 Dichlorphenol - Indophenollosung wurden in das Paremchym der Tonsillen eingespritzt und die Zeit bis zur Entfärbung des Farbstoffes gemessen Die Anwendung von Indophenol ist insofern vorteilhaft als die Messung nur in kurzer Zeit moglich ist Beide Prufungen haben sogar zur Differentialdiagnose zwischen der einfachen Tonsillenhypertrophie und der chronischen Entzundung eine ausschlaggebende Bedeutung Die Werte uber 5 Stunden bei Methylenblau und uber 5 Minuten bei Indophenol stellen die Grenze zur Bestimmung dar Bei Tonsillen der Fokal Infektion war die Entfärbungszeit wesentlich länger als bei Tonsillitis chronica simplex

4 *Elektrodiagnose* Die Potenzialdifferenz wurde nun mit Hilfe eines Messapparates von dem Ruhestrom und zwar mit der Depolarisationselektrode, gemessen Es erwies sich aber dass diese Methode als eine Herd-Diagnostik kaum wertvoll ist

IV Lokale Herd Provokation

1 *Tonsillen Massage* Mit einem von uns konstruierten Tonsillen Massagegerät wurde je 5 Minuten beiderseits der lokale Reiz gegeben und der Befund nach der Provokation zeitlich verfolgt Da die Veränderung der Körperwärme erst nach 15 Minuten und die Veränderung der Leukozytenzahl sowie des Blutsenkungswertes erst nach 3 Stunden am deutlichsten waren so wurden diese Werte nunmehr als Grundlage zur Messung gewählt Als Kriterien für den positiven Ausfall wurden diejenige Werte die über den höchsten Wert der Veränderungen bei Tonsillitis chronica simplex weit hinausgehen und zwar für die Körpertemperatur die Erhöhung um 0 55 C für die Leukozytenzahl die Zunahme um 1200 und für den Senkungswert die Beschleunigung um mehr als 12 mm als am geeignesten betrachtet Bei der Tonsillitis chronica simplex blieb die Veränderung der Werte immer innerhalb dieses Raumes Bei tonsillogenen Fokal Infektionen war aber die Leukozytenzahl in 53 3 % positiv wobei die Zunahme über 2000 in 40 % zu beobachten war Daher ist die Bestimmung der Leukozytenzahl besonders wertvoll Die Senkungsreaktion war dabei in 30 8 % positiv Die Veränderung war aber bei Rheumatismus besonders gross Bei Subfebrilität und Nephritis war die Senkungsreaktion in der Hälfte der Fälle des Rheumatismus positiv Die Körpertemperatur war aber nur in 20 % positiv Jedoch war das Resultat nach der Tonsillektomie sehr gut Die Veränderung von Urin war bei Nephritis ganz auffallend und zwar war die Zunahme von Eiweiss und Erythrozyten unausbleiblich Bei 14 Fällen der Tonsillitis chronica focalis bei denen aber mindestens 2 von 3 Kennzeichen d. h Korperpemperatur Leukozytenzahl und Blutkörperchensenkung positiv waren wurden die Veränderungen durch lokale Massage in 1 bis 6 Monaten nach der Tonsillektomie sämtlich negativ Da die sekundären Erkrankungen dadurch sämtlich zur Heilung kamen so hat die Tonsillenmassage eine grosse diagnostische Bedeutung

2 *Provokation durch Ultrakurzwellen* Die biologische Wirkung der Ultrakurzwellen ist mannichfaltig Physikalisch ist aber die Wärmewirkung der Ultrakurzwellen als molekulare Elektromassage anzusehen Durch diese Heilmethode wird aktive Hyperämie hervorgerufen und gleichzeitig auch die Verstärkung der Lymphströmung bedingt Infolgedessen wird die Resorption der krankhaften Produkte *in loco beschleunigt* *Als Wirkungsmachanismus* wird angenommen dass die vom Primärherd frei gewordenen Bakterien oder Toxine aber auch Eiweissbestandteile des Gewebes oder Herd Antigenen dadurch beeinflusst werden Die Reaktion durch indirekte 5 Minuten Bestrahlung der Mandeln von dem Seitenhals her ist aber nur schwach und unzulänglich Die indirekte Bestrahlung für 10 und 15 Minuten zeigt dagegen einen fast gleichen Erfolg so dass angenommen werden kann dass die 10 Minuten Bestrahlung schon genügend ist

Wenn wir nun nach dem Kriterium für die positive Tonsillen Massage die Resultate dieser beiden Methoden betrachten so ist die Veränderung der Temperatur nach 15 Minuten gross und bei Tonsillitis chronica focalis in zirka 52 % positiv wobei die Beeinflussung aber nur innerhalb von 45 Minuten geblieben ist Die Leukozytenzahl war nach 3 Stunden in 58 6 % positiv Dabei

perior bzw. Lacunen eingeschoben und damit die Radioaktivität der Mandel, des Blutes und des Urins bestimmt. Die Resorptionsfähigkeit der Mandel zeigte dabei in 1—2 Stunden nach der Einführung den maximalen Wert, so dass dieser Zeitraum nunmehr zur Grundlage der Bestimmung gewählt wurde. Die Radioaktivität war aber bei normalen Tonsillen nur in 22 % der Fälle und bei Tonsillitis chronica simplex auch in 36.3 % bei der Tonsillitis chronica focalis, besonders aber bei Nephritis, in 65.9 % nachweisbar. Ferner war die Aktivität bei Subfebrilitäten in 40.6 % und bei Rheumatismus jedoch nur in 18.4 % feststellbar. Die Radioaktivität des Blutes, die nach 3 Stunden ihren grossten Wert zeigte, war bei Tonsillitis chronica simplex etwas grosser als bei normalen Tonsillen. Bei Tonsillitis chronica focalis, und zwar bei Rheumatismus, bei dem die Resorption aus der Mandel verzögert ist, und die Ausscheidung durch den Urin noch normal ist, zeigte die Radioaktivität nach 4 Stunden ihren maximalen Wert. Dabei war jedoch C.P.M. nicht so gross. Bei Nephritis, bei der die Resorption noch günstig, jedoch die Ausscheidung beeinträchtigt ist, war die Radioaktivität gross und dauerte sogar noch längere Zeit. Die Radioaktivität des Urins erreichte, wie die des Blutes, gegenüber der subcutanen Injektion erst 1—2 Stunden später ihren maximalen Wert. Bei der Nephritis war die Radioaktivität des Urins aber gering und es wurde sogar eine Verzögerung festgestellt. Die Prüfung über den positiven Prozentsatz, indem 2 Kennzeichen, d. h. die Resorption aus Mandellacunen über 30 % und im Blut über 150 C.P.M. positiv waren, ergab, dass der totale Prozentsatz bei Tonsillitis chronica simplex nur 25 %, dagegen bei Tonsillitis chronica focalis, besonders aber bei Nephritis, 75 % und bei der Subfebrilität 62.5 % betrug, während er bei Rheumatismus nur 14.3 % zeigte, also niedrig war. Da die Erfolgsziffer nach Tonsillektomie bei positiven Fällen sehr gross war: 91.7 %, dagegen bei negativen Fällen viel kleiner war: 41.7 %, so ist die Erfassung des Charakteristikum der sekundären Erkrankungen diagnostisch auch bedeutungsvoll.

3. *Farbstoff-Entfärbungsreaktion in Tonsillen.* Je 0.2 cc einer 0.8 %igen Methylenblaulösung oder einer 1 %igen 2—6 Dichlorphenol-Indophenollösung wurden in das Pareimchym der Tonsillen eingespritzt und die Zeit bis zur Entfärbung des Farbstoffes gemessen. Die Anwendung von Indophenol ist insofern vorteilhaft, als die Messung nur in kurzer Zeit möglich ist. Beide Prüfungen haben sogar zur Differentialdiagnose zwischen der einfachen Tonsillenhypertrophie und der chronischen Entzündung eine ausschlaggebende Bedeutung. Die Werte über 5 Stunden bei Methylenblau und über 5 Minuten bei Indophenol stellen die Grenze zur Bestimmung dar. Bei Tonsillen der Fokal-Infektion war die Entfärbungszeit wesentlich länger als bei Tonsillitis chronica simplex.

4. *Elektrodiagnose.* Die Potenzialdifferenz wurde nun mit Hilfe eines Messapparates von dem Ruhestrom, und zwar mit der Depolarisationselektrode, gemessen. Es erwies sich aber, dass diese Methode als eine Herd-Diagnostik kaum wertvoll ist.

IV Lokale Herd Provokation

1 *Tonsillen Massage* Mit einem von uns konstruierten Tonsillen Massagegerät wurde je 5 Minuten beiderseits der lokale Reiz gegeben und der Befund nach der Provokation zeitlich verfolgt Da die Veränderung der K r perwärme erst nach 15 Minuten und die Veränderung der Leukozytenzahl sowie des Blutsenkungswertes erst nach 3 Stunden am deutlichsten waren so wur den diese Werte nunmehr als Grundlage zur Messung gewählt Als Kriterien fi r den positiven Ausfall wurden diejenige Werte die über den h chsten Wert der Veränderungen bei Tonsillitis chronica simplex weit hinausgehen und zwar fur die K rpertemperatur die Erh hung um 0 55 C fur die Leukozytenzahl die Zunahme um 1200 und fi r den Senkungswert die Beschleunigung um mehr als 12 mm als am geeignesten betrachtet Bei der Tonsillitis chronica simplex blieb die Veränderung der Werte immer innerhalb dieses Raumes Bei tonsil logenen Fokal Infektionen war aber die Leukozytenzahl in 53 3 % positiv wobei die Zunahme über 2000 in 40 % zu beobachten war Daher ist die Bes timmung der Leukozytenzahl besonders wertvoll Die Senkungsreaktion war dabei in 30 8 % positiv Die Veränderung war aber bei Rheumatismus beson ders gross Bei Subfebrilität und Nephritis war die Senkungsreaktion in der Hälfte der Fälle des Rheumatismus positiv Die K rpertemperatur war aber nur in 20 % positiv Jedoch war das Resultat nach der Tonsillektomie sehr gut Die Veränderung von Urin war bei Nephritis ganz auffallend und zwar war die Zunahme von Eiweiss und Erythrozyten unausbleiblich Bei 14 Fällen der Tonsillitis chronica focalis bei denen aber mindestens 2 von 3 Kennzei chen d h K rperpemperatur Leukozytenzahl und Blutk rperchensenkung positiv waren wurden die Veränderungen durch lokale Massage in 1 bis 6 Monaten nach der Tonsillektomie sämtlich negativ Da die sekundären Erk rankungen dadurch sämtlich zur Heilung kamen so hat die Tonsillenmassage eine grosse diagnostische Bedeutung

2 *Provokation durch Ultrakurzwellen* Die biologische Wirkung der Ul trakurzwellen ist mannichfaltig Physikalisch ist aber die Wärmewirkung der Ultrakurzwellen als molekulare Elektromassage anzusehen Durch diese Heilmethode wird aktive Hyperämie hervorgerufen und gleichzeitig auch die Verstärkung der Lymphstr mung bedingt Infolgedessen wird die Resorption der krankhaften Produkte in loco beschleunigt. Als Wirkungsmachanismus wird angenommen dass die vom Primärherd frei gewordenen Bakterien oder Toxine aber auch Eiweissbestandteile des Gewebes oder Herd Antigenen da durch bee nflusst werden Die Reaktion durch indirekte 5 Minuten Bestra hlung der Mandeln von dem Seitenhals her ist aber nur schwach und unzulän glich Die indirekte Bestrahlung fur 10 und 15 Minuten zeigt dagegen einen fast gleichen Erfolg so dass angenommen werden kann dass die 10 Minuten Bestrahlung schon gen gend ist.

Wenn wir nun nach dem Kriterium f ir die positive Tonsillen Massage die Resultate dieser beiden Methoden betrachten so ist die Veränderung der Tem peratur nach 15 Minuten gross und bei *Tonsillitis chronica focalis* in zirka 52 % positiv wobei die Beeinflussung aber nur innerhalb von 45 Minuten geblie ben ist. Die Leukozytenzahl war nach 3 Stunden in 58 6 % positiv *Dabei*

perior bzw. Lacunen eingeschoben und damit die Radioaktivität der Mandel des Blutes und des Urins bestimmt Die Resorptionsfähigkeit der Mandel zeigte dabei in 1—2 Stunden nach der Einführung den maximalen Wert so dass dieser Zeitraum nunmehr zur Grundlage der Bestimmung gewählt wurde Die Radioaktivität war aber bei normalen Tonsillen nur in 22 % der Fälle und bei Tonsillitis chronica simplex auch in 36 3 % bei der Tonsillitis chronica focalis besonders aber bei Nephritis in 65 9 % nachweisbar Ferner war die Aktivität bei Subfebrilitäten in 40 6 % und bei Rheumatismus jedoch nur in 18 4 % feststellbar Die Radioaktivität des Blutes die nach 3 Stunden ihren grossten Wert zeigte war bei Tonsillitis chronica simplex etwas grosser als bei normalen Tonsillen Bei Tonsillitis chronica focalis und zwar bei Rheumatismus bei dem die Resorption aus der Mandel verzögert ist und die Ausscheidung durch den Urin noch normal ist zeigte die Radioaktivität nach 4 Stunden ihren maximalen Wert Dabei war jedoch C P M nicht so gross Bei Nephritis bei der die Resorption noch günstig jedoch die Ausscheidung beeinträchtigt ist war die Radioaktivität gross und dauerte sogar noch längere Zeit. Die Radioaktivität des Urins erreichte wie die des Blutes gegenüber der subcutanen Injektion erst 1—2 Stunden später ihren maximalen Wert Bei der Nephritis war die Radioaktivität des Urins aber gering und es wurde sogar eine Verzögerung festgestellt Die Prüfung über den positiven Prozentsatz indem 2 Kennzeichen d h die Resorption aus Mandellacunen über 30 % und in Blut über 150 C P M positiv waren ergab dass der totale Prozentsatz bei Tonsillitis chronica simplex nur 25 % dagegen bei Tonsillitis chronica focalis, besonders aber bei Nephritis 75 % und bei der Subfebrilität 62 5 % betrug während er bei Rheumatismus nur 14 3 % zeigte also niedrig war Da die Erfolgsziffer nach Tonsillektomie bei positiven Fällen sehr gross war 91 7 % dagegen bei negativen Fällen viel kleiner war 41 7 % so ist die Erfassung des Charakteristikum der sekundären Erkrankungen diagnostisch auch bedeutungsvoll

3 *Farbstoff Entfärbungsreaktion in Tonsillen* Je 0 2 cc einer 0 8 %igen Methylenblaulösung oder einer 1 %igen 2—6 Dichlorphenol - Indophenollösung wurden in das Paremchym der Tonsillen eingespritzt und die Zeit bis zur Entfärbung des Farbstoffes gemessen Die Anwendung von Indophenol ist insofern vorteilhaft als die Messung nur in kurzer Zeit möglich ist Beide Prüfungen haben sogar zur Differentialdiagnose zwischen der einfachen Tonsillenhypertrophie und der chronischen Entzündung eine ausschlaggebende Bedeutung Die Werte über 5 Stunden bei Methylenblau und über 5 Minuten bei Indophenol stellen die Grenze zur Bestimmung dar Bei Tonsillen der Fokal Infektion war die Entfärbungszeit wesentlich länger als bei Tonsillitis chronica simplex

4 *Elektrodiagnose* Die Potenzialdifferenz wurde nun mit Hilfe eines *Messapparates von dem Ruhestrom* und zwar *mit der Depolarisationselektrode*, gemessen Es erwies sich aber dass diese Methode als eine Herd-Diagnostik kaum wertvoll ist

unigung der Senkungsreaktion um 10 mm und daruber nachweisbar sind so wurde dieses als positiv bezeichnet Es ergab sich dass sowohl die Korpertemperatur wie auch die Leukozytenzahl in 75 % und die Senkungsreaktion in 59 4 % positiv sind und dass der positive Prozentsatz bei der direkten Bestrahlung sogar viel hoher als bei der indirekten Bestrahlung ist und daruber hinaus lasst sich durch die direkte Methode die Tonsillitis chronica focalis ganz scharf von der Tonsillitis chronica simplex unterscheiden Bei positiven Fallen war die Erfolgsziffer nach der Tonsillektomie sehr hoch wie z B die Korpertemperafur und Leukozytenzahl je 91 7 % und die Blutsenkung sogar 100 % sind Es lasst sich also mit Recht sagen dass die direkte Bestrahlung eine bestbewahrte Diagnostik ist

3 *Rontgen Provokation* Zur Rontgen Provokation hat DRIAK 5 r der biologischen Strahlendosis verwand Wir haben aber beiderseits mit je 30 r bestrahlt Bei der Tonsillitis chronica focalis waren die Zunahme der Leukozytenzahl uber 1000 in 71 4 % und die Beschleunigung der Senkungsreaktion uber 5 mm in 63 % zu beobachten Ferner trat die Harnveranderung bei der Nephritis in 78 % auf Rheumatismus mit der Verstarkung oder Erleichterung des Schmerzens kam nach der Tonsillektomie zur Heilung Deshalb ist die Beobachtung des Schmerzes auch wichtig Die durch diese Methode provozierte Reaktion ist aber nicht sehr signifikant jedoch insofern charakteristisch da diese Methode nur wenig die Tonsillitis chronica simplex beeinflusst

4 *Galvanisatorische Provokation* MARINO hat die Mandel mit 5 mA 5 Minuten lang galvanisatorisch gereizt Die Methode hat aber abgesehen von der tonsillogenen Nephritis nur geringen Wert

5 *Niederfrequenz-Provokation* Die Methode wurde bisher noch von niemandem probiert Wir haben nun eine *Oral Elektrode hergestellt und* damit die Mandel beiderseits bei 30 cycle 3 mA je 5 Minuten gereizt Da die Abweichung von der Tonsillitis chronica simplex immer grosser wurde so haben wir folgende 2 Kennzeichen fur den positiven Ausfall als zweckentsprechend erdacht Zunahme der Leukozytenzahl nach 1 Stunde uber 1000 und Beschleunigung der Blutsenkung nach 3 Stunden uber 5 mm Positive Falle dieser 2 Kennzeichen wurden aber bei Tonsillitis chronica simplex noch nicht dagegen bei Tonsillitis chronica focalis in 38 7 % beobachtet Die Reaktion war jedoch nicht so auffallend wie bei der Ultrakurzwellen Provokation *Ausserdem noch hat die Methode den Nachteil da die Patienten wahrend der* Reizung geringe Schmerzen verspuren Es sei hier nur ganz kurz darauf hingewiesen dass die *Ultraschall Provokation* gar nicht geeignet ist.

6 *Hyaluronidase-Test* Zur Diagnose wurde die Hyaluronidase aus dem Grund verwendet da das Ferment fahig ist *die Hyaluronsaure d h. die* Grundsubstanz der Bindegewebe zu hydrolysieren und dadurch die Permeabilitat der Gewebe zu erh hen dann aber die Diffusion der Bakterien damit auch die Infektion zu begunstigen und sogar die Resorption der Antigene zu f rdern BALTALENA und RINALDI haben die Hyaluronidase bei atiologisch unbekannten Subfebrilitaten verwendet und diejenigen Falle als positiv bezeichnet, bei denen sich die K rpertemperatur erh ht. Bei uns wurden 2000 V U Hyaluronidase in 1 cc der physiologischen Kochsalzl sung gel st und davon je 0 5 cc in Parenchym der beiden Tonsillen injiziert Die durchschnittliche orale Temperatur zeigte aber bei normalen Tonsillen keine Veranderung j ch

traten Plasmazellen und Eosinophilzellen auch vermehrt auf Die Veränderung von BSG war nach 3 Stunden deutlich und zwar in ca 45 % positiv Die Veränderung des Urins war bei Nephritis deutlich deswegen auch bedeutungs voll Bei Fällen der Tonsillitis chronica focalis bei denen mindestens 2 von 3 Kennzeichen d h Körpertemperatur Leukozytenzahl und BSG positiv aus fielen wurde nach der Tonsillektomie die örtliche Provokation vorgenommen die jedoch in sämtlichen Fällen negativ war Da bei diesen Fällen die sekun dären Krankheiten sämtlich geheilt worden sind so ist das Urteil nach dieser Methode am besten vertrauenswürdig

Wir haben nun ausser der indirekten Bestrahlungsmethode noch eine direkte Bestrahlungsmethode ersonnen die wie folgt ausgeführt wird Dem Ultra *kurzwellen-Aussender wurde ein Dekrement-Regulator angesetzt* *Andererseits* wurden Mandel Elektroden hergestellt Eine Elektrode der Gegenseite wurde mit dem Akryl Band am Seitenhals fixiert um damit die direkte Bestrahlung vorzunehmen (Abb 6 und 7)

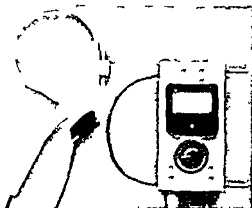


Abb 6 Tonsillen Elektrode und Dekrement Regulator (NOSAKA)



Abb 7 Direkte Provokation durch Ultrakurzwellen

Wenn unter Bestrahlungsbedingungen von je 5 Minuten beiderseits der Ton sillen und bei gleichbleibender Messungszeit wie oben die Erhöhung der Tem peratur um 0 45 C die Zunahme der Leukozytenzahl um 1000 und die Beschle

unigung der Senkungsreakt on um 1 mm und darüber nachwe wurde dieses als positiv bezeichnet. Es ergab ch da s sowohl d. peratur wie auch die Leukozytenzahl in 75 ₀ und die Senkung 59 4 % positiv sind, und dass der po tive Prozentsatz bei der d. rahlung sogar viel h her als bei der indirekten Be trahlung ist. hinaus lasst sich durch die direkte Methode die Tonsillitis chr ganz scharf von der Ton illitis chron ca simplex untersche den Fällen war die Erfolgsziffer nach der Tonsillektomie sehr hoch, v Korpertemperafur und Leukozytenzahl je 91 7 %, und die Blutse 100 % sind. Es lasst sich also mit Recht sagen dass die direkte eine bestbewahrte Diagnostik ist.

3. *Röntgen Provokation* Zur R ntgen Provokation hat Di biologischen Strahlendosis verwand Wir haben aber beiderseit bestrahlt. Bei der Tonsillitis chronica focalis waren die Zunahm zytenzahl uber 1000 in 71 4 % und die Beschleunigung der Senk uber 5 mm in 63 % zu beobachten Ferner trat die Harnverand Nephritis in 78 % auf Rheumatismus mit der Verstarkung ode ung des Schmerzens kam nach der Tonsillektomie zur Heilung die Beobachtung des Schmerzes auch wichtig Die durch diese vozierte Reaktion ist aber nicht sehr signifikant, jedoch insofern stisch, da diese Methode nur wenig die Tonsillitis chronica implex

4 *Galvanisatorische Provokation* MARINO hat die Mandel Minuten lang galvanisatorisch gereizt. Die Methode hat aber al der tonsillogenen Nephritis nur geringen Wert.

5 *Niederfrequenz Provokation* Die Methode wurde bish niemandem probiert. Wir haben nun eine Oral Elektrode he damit die Mandel beiderseits bei 30 cycle 3 mA je 5 Minuten die Abweichung von der Tonsillitis chronica simplex immer grss haben wir folgende 2 Kennzeichen fur den positiven Ausfall als z chend erdacht Zunahme der Leukozytenzahl nach 1 Stund und Beschleunigung der Blutsenkung nach 3 Stunden uber 5 m Fälle dieser 2 Kennzeichen wurden aber bei Ton illitis chronica nicht, dagegen bei Tonsillitis chronica focalis in 35 7 % beobachte ktion war jedoch nicht so auffallend wie bei der Ultrakurzwellen Ausserdem noch hat die Methode den Nachteil da die Patienten Reizung geringe Schmerzen ver puren Es sei hier nur ganz hingewiesen dass die *Ultraschall Provokation* gar nicht geeignet

6 *Hyaluronidase-Test* Zur Diagno e wurde die Hyaluroni Grund verwendet da das Ferment fähig ist die Hyaluronsäu Grundsubstanz der Bindegewebe zu hydrolysieren und dadurch bilität der Gewebe zu erh hen dann aber die Diffu ion der Bak auch die Infektion zu begunstigen und sogar die Resorption der f rdern BALTALENA und RINALDI haben die Hyaluronidase b unbekannten Subfebrilitäten verwendet und diejenigen Fälle als chnet bei denen sich die K rpertemperatur erh ht. Bei uns wu U Hyaluronidase in 1 cc der physiologischen Kochsalzl sung gelö je 0 cc in Parenchym der beiden Tonsillen injiziert. Die dur orale Temperatur zeigte aber bei normalen Tonsillen keine Veränd

leichter Gelenkschmerz beobachtet

3 *Ganslmayer-Test* Antisepton 800 enthalt hauptsachlich die Vakzine der abgetoteten Streptokokken Staphylokokken Colibazillen Pyozyaneusbazillen usw und daneben noch antibakterielle und antitoxische Sera Bei diesem Test handelt es sich um eine Antigen Antikorper Reaktion indem die Vakzine d h Antigene mit dem in der Abwehrzone des Krankheitsherdes fixierten Antikorper und dazu noch die Antisera mit den im Krankheitsherd befindlichen Bakterien und Toxinen immunbiologisch reagieren

Die Pruefung ergab dass der positive Prozentsatz bei Tonsillitis chronica simplex ahnlich wie bei sekundaren Erkrankungen der Fokal-Infektion nur 21 4 % jedoch bei Rheumatismus tonsillogenen Ursprunge so hoch wie 77 8 % also diagnostisch bedeutungsvoll ist Da aber die Erfolgsziffer nach der Tonsillektomie bei positiven Fallen etwas kleiner als bei negativen Fallen war so durfte dieser Test diagnostisch nicht hoch geschatzt werden Die Reaktion trat dabei nach 2—8 Stunden auf Die Nebenwirkung war wenn auch etwas geringer als beim Spenglersan Test denn noch in zirka 1/4 der Falle als harmlose 3—4 Tage lang dauernde Hautr tung oder Induration wahrnehmbar

4 *Pyrifer Test* Pyrifer ist eine Aufschwemmung der abget teten Coli Bazillen Wird eine geringe Menge von Pyrifer injiziert so werden die noch latenten Entzudungsprozesse provoziert wobei sich die K rpertemperatur allmahlich erh ht Dann wird beobachtet dass die Schmerzreaktion subjektiv und objektiv im Krankheitsherd auftritt

Allgemeinreaktion nach der Original Vorschrift mit 0 3 cc war aber zu stark so dass wir fur Manner 0 1 cc von 5 Einheiten und fur Frauen 0 08 cc von 4 Einheiten als geeignete Dosis wahlen Die Reaktion trat bei Tonsillitis chronica simplex in 21 4 % und bei Tonsillitis chronica focalis aber in 73 1 % positiv auf Die Erfolgsziffer nach der Tonsillektomie bei positiven Fallen betrug 89 5 % war also gunstig Die K rpertemperatur erh hte sich unausbleiblich und zwar fing sie 2 Stunden nach der Injektion allmahlich an zusteigen und erreichte nach ungefahr 12 Stunden ihren h chsten Wert um nach ca 20 Stunden wieder zur Norm zuruckzukehren Als Nebenwirkung war der mit dem Fieber einhergehende Kopfschmerz unfehlbar auch wurden Gelenkschmerz Lenden und Ruckenschmerz in der Halfte der Falle beobachtet

Werden nun oben erwahnte 3 diagnostisch Methoden im Hinblick auf die [illegible]ostische Treffsicherheit und die Prognose der tonsillektomierten sekun[illegible] Krankheiten miteinander verglichen so bewahren sich die Methoden in der [illegible]ihenfolge Pyrifer Spenglersan Antisepton Vor allem steht die letzte Methode ganz zuruck Aber diese 3 immunbiologischen Methoden haben die gemeinsame Schwachheit, dass das Urteil der Resultate hauptsachlich nach der Angabe der Patienten gemacht wird und die Nebenwirkung ziemlich stark ist

5 *Antistreptolysin Test* Aus dem Grunde dass antigenaktive Hamolysine in hamolytischen Streptokokken vorhanden sind hat man auch auf die Bestimmung des Antik rper Titers der hamolytischen Streptokokken im Serum von den mit diesem Erreger infizierten Patienten grosses Gewicht gelegt um dadurch die Diagnose der Infekten oder der mit hamolytischen Streptokokken aufs engste zusammenhangenden Herd Infektion fest zu stellen

Bei der Tonsillitis chronica simplex zeigte AST in Serum durchschnittlich

194 Einheiten (ueber 500 Einheiten noch nicht), die grosser ... bei normalen Tonsillen Bei Tonsillitis chronica focalis wa... Einheiten, also bei weitem grosser Beim Fehlen anderer H... die Fokal-Infektion sehr verdächtig Im allgemeinen ist der T... Einheiten als normale Obergrenze angenommen worden, so dass ... 250 Einheiten nunmehr als positiv betrachtet werden kann

Nach diesen Kriterien war der positive Prozentsatz bei Ton... simplex 35 5 % und bei Tonsillitis chronica focalis 57 1 % De... entsprach gerade der prozentualen Nachweisziffer der hämolyt... kokken aus Tonsillen

Der durchschnittliche Titer von AST wurde bei Tonsillitis chr... nach der Tonsillektomie nur geringfugig jedoch bei positiven Fa... sillektomie nach der Operation bis zu 57 % herabgesetzt Der Grad... zung war aber bei Tonsillitis chronica focalis noch grosser und ... der Herabsetzung in 88 % auch grosser

Hierauf haben wir nun den Einfluss der Provokation auf A... Ultrakurzwellen- und Hyaluronidase-Massagentest beobachtet, d... nicht zu diesem Zweck verwendet worden ist Es ergab sich, d... Anstieg durch Provokation aber bei Tonsillitis chronica simplex... und 25 % war, wahrend er bei Tonsillitis chronica focalis je 70... %, also sehr deutlich war Die Erfolgsziffer nach der Tons... bei positiven Provokationsfallen beinahe 90 %, also viel höher a... einfachen positiven Fallen, was der Differentialdiagnose-Stellung... chronica simplex und focalis oder zur Indikationsstellung der ... als Merkmal dienen kann Dabei muss aber der grosste Wert ... Anstieg durch Provokation gelegt werden

6 *Antistaphylolysin Test* In weitaus der Mehrzahl der... Tonsillitis chronica simplex war der Titer Anstieg nur unterhal... heit Die Tonsillitis chronica focalis umfasste aber Falle m... wobei der Titer bei Nephritis hoch und bei Rheumatismus jedoc... Deshalb ist dem Antistaphylolysin Test kein diagnostischer Wert... wie dem AST

7 *Latex-Fixationstest C-reaktives Protein (CRP) Test u... Fokaltest* Der Latex Fixationstest war bei Tonsillitis chron... gativ dagegen bei Tonsillitis chronica focalis in 31 6 % po... wurden durch Provokation des positiven Umschlages der Reak... Verstarkung ungefahr in 58 % beobachtet, besonders war si... Rheumatismus Jedoch ist der diagnostische Wert dieser Test... schätzen

CRP-Test fiel nur bei Tonsillitis chronica focalis positiv a... Tonsillektomie wurde der Test in hohem Ausmasse, d.h. in 88... Die Erfolgsziffer der Tonsillektomie ist also ziemlich hoch... kommt diesem Text bei positiven Fällen eine gewisse Bedeu... Test besitzt aber einen schwachen Punkt, da er bei dem son... diagnostische Treffsicherheit verzeichneten Rheumatismus nur... ausfiel Der Penicillin Test war jedoch beim Tonsillen-Herd s...

VI Annulations- und Kombinationsdiagnose

1 *Impletol-Test* Bei diesem Test handelt es sich um eine Heil Anästhesie, in der nämlich zur Beobachtung des Abklingens der neuralgischen Schmerzen usw Impletol ein Neuraltherapeutikum in den verdächtigen Krankheitsherd eingespritzt wird Die Methode wird wie folgt durchgeführt Je 1 cc von Impletol wird durch den vorderen Gaumenbogen beiderseits in Submukosa von 2 oberen Polen der Tonsillen injiziert um dadurch Quadeln zu bilden

Durch diese Methode wurde der Schmerz von Gelenkrheumatismus scheinbar tonsillogenen Ursprungs in 82.1 % gebessert Bei Gelenkrheumatismus offenbar tonsillogenen Ursprungs betrug die diagnostische Treffsicherheit sogar 96.9 % Nach der Tonsillektomie bei diesen positiven Fällen wurde 85 % zur Heilung gebracht Der positive Prozentsatz der erfolgreich Behandelten war vor der Operation auch so sehr hoch wie 94.1 % (Tabelle 2) Die gleich nach der Injektion bis zur Schmerzlinderung erforderliche Zeit betrug meist 4 Stunden Wohl aber trat der Erfolg bei auffallend tonsillogenen Fällen schon innerhalb von 3 Stunden ein (Abb 9)

TABELLE 2 *Resultate von Impletol Test (Bei [illegible] Fällen sehr wirksam und bei 45 Fällen wirksam)*

einzelne Gruppe	tonsillogen	pseudotonsillogen	tosillogen allgemein	tonsillogen unklar	Begleitung von sekundären Khten	Erfolgsziffer d Tonsillektomie bei positiven Fällen	Positiver Prozentsatze bei erfolgreicher Tonsillektomie
Fälle	32	35	67	32	99	40	36
erfolgreiche Fälle	31	24	55	11	66	34	34
%	96.9	68.6	82.1	34.4	66.7	85.0	94.4

Wir haben bisher jedoch noch nicht solche sekundäre Phänomenen beobachtet bei denen der Schmerz wie HUNEKE darauf hinwies sofort in 100 % zum Abklang kam Die Dauer der Schmerz Linderung betrug bei uns am kürzestens 4 Stunden und am längsten 4 Tage durchschnittlich 29.5 Stunden Die Dauer war beim Fall auffallend tonsillogenen Ursprungs länger und beim Fall der unklaren Ätiologie kürzer Bei fortgesetzter Injektion wurden solche Fälle beobachtet in denen die schmerzlindernde Dauer trotz zunehmender Anzahl der Injektionen nicht abgekürzt werden konnte Solche Fälle waren bei tonsillogenen Erkrankungen sehr häufig in 93.5 % dagegen bei ätiologisch unklaren Krankheiten wenig häufig wie in 54.5 % zu beobachten Es ist notwendig dass die Injektion mindestens 3 mal vorgenommen werden muss (Abb 10)

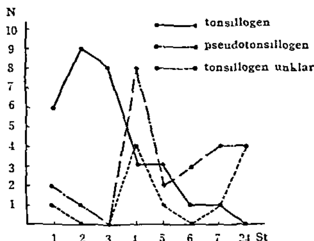


ABB 9 Die bis zum Abklingen des Gelenkschmerzens erforderliche Zeit

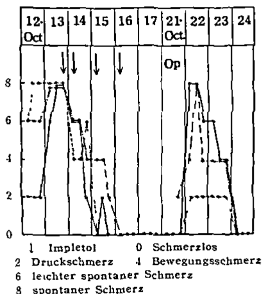


ABB 10 Einfluss der Impletol-Injektion und der Tonsillektomie auf den Schmerz

Der Erfolg ist unabhängig von den erkrankten Jahren des Rheumatismus, und tritt bei der platten Form der Mandel am häufigsten und, je kleiner der Vorsprung der Mandel ist, desto häufiger auf, was unsere Annahme über die Morphologie der rheumatischen Tonsillen belegen darf

Auch die Gelenkanschwellung, die bisher nur wenig berücksichtigt worden war, wurde von 23 Fällen bei 9 zur Abschwellung und bei 5 zum Verschwinden

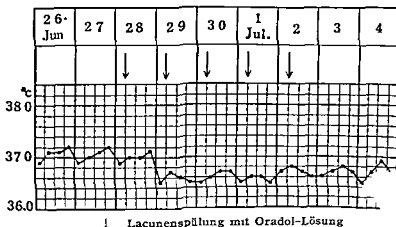
gebracht. Ferner waren die Schmerzen durch Impletol-Iontophorese in ca 67 % wesentlich herabgemindert

Was den Wirkungsmechanismus anbelangt, lässt sich Folgendes erklären Procain blockiert wahrscheinlich synergetisch mit Koffein die aus dem Fokus über Nerven- und Gefäss-System und weiterhin bis zum Zwischenhirn zur organisierten Umstimmung führenden Impulse

Die positiven Fälle im Impletol-Test wurden nach der Tonsillektomie in 85 % zur Heilung gebracht Der positive Prozentsatz bei erfolgreichen tonsillektomierten Fällen war sehr hoch und betrug 94 4 %. Dieser Test stellt also ein unentbehrliches und hervorragendes Verfahren zur Herd-Diagnose der Tonsillen bei rheumatischen Erkrankungen und zugleich zur Behandlung dar Ferner kann der positive Ausfall als Indikator zur Tonsillektomie dienen Es ist auch von grossem Belang, dass die Dauer bis zum Verschwinden des Schmerzens nur innerhalb von 3 Stunden bleibt und die Dauer der Schmerz Linderung durch fortgesetzte Injektionen keinerlei verkürzt wird

2 *Spülung der Tonsillen-Lacunen* Durch Spülung der Tonsillenlacuna besonders aber der Lacuna superior, werden Keim-haltige Eiterpfropfe bzw Detritusmassen völlig entfernt und der Einfluss auf die daraus stammenden Fernsymptome beobachtet. Die Methode wird besonders zur Diagnose der chronischen Subfebrilität verwendet Der Wirkungsmechanismus wird so erklärt, dass einerseits die Resorption der Bakterien und der produzierten Toxine, aber auch der Antigen-haltigen Substanzen ausgeschaltet werden, und andererseits nach dem gebesserten Befund von EKG auch die Beteiligung des autonomen Nervensystems geschätzt wird

Mit Hilfe eines Lacunenspülungsgerätes, und zwar mit 20 cc der Oradol-, Leukomycin, Cathocillin-Lösung usw, werden die Lacunen täglich während 5—7 Tage gespült Dabei tritt die Entfieberung bei tonsillogenen Subfebrilitäten in einem sehr hohen Prozentsatz wie 95 5 % ein Anzeichen der Entfieberung kann in der Überzahl der Fälle schon nach 2 maligen Spülungen beobachtet werden (Abb 11)



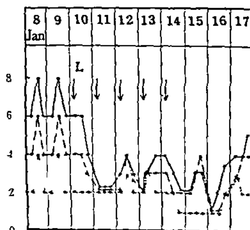
↓ Lacunenspülung mit Oradol-Lösung

Abb 11 Entfieberung durch Lacunen-Spülungen

Die Veränderung der Leukozytenzahl und der Blutsenkung war aber nicht deutlich Die Beschwerde bei Rheumatismus war durch 5 malige Spulungen in 66.7 % gebessert, ebenso war der Harnbefund bei Nephritis in 7 von 10 Fallen gebessert (Tabelle 3 und Abb 12)

TABELLE 3. *Resultate der Lacunenspulung von Tonsillen*

	Krankheiten	Positiver Prozentsatz der Reaktion: Tonsillogen	Pseudotonsillogen	tonsillogen allgemein	tonsillogen unklar	nicht tonsillogen	Erfolgsziffer d Tonsillektomie bei positiven Fallen	Erfolgsziffer d Tonsillektomie bei negativen Fallen	Positiver Prozentsatze bei erfolgreicher Tonsillektomie
Tonsillogene Erkrankungen	Rheuma	4/5 80.0	2/4 50.0	6/9 66.7	0/4 0	0/1 0	4/4 100.0	1/2 50.0	4/5 80.0
	Nephritis	5/5 100.0	2/5 40.0	7/10 70.0	0/3 0		5/5 100.0	0/3 0	5/5 100.0
	Subfebrilitat	17/18 94.4	4/4 100.0	21/22 95.5	0/6 0	0/1 0	19/19 100.0	1/1 100.0	19/20 95.0
	Herzleiden		1/3 33.3	1/3 33.3			1/1 100.0	1/2 50.0	1/2 50.0
	insgesammt	26/28 92.9	9/16 56.3	35/44 79.5	0/13 0	0/2 0	29/29 100.0	3/8 37.5	29/32 90.6



L Lacunenspulung mit Cathocillin V-Lösung
•——• Arm •– –• Schenkel • • Hufte

ABB 12 Milderung der Schmerzen durch Spulung der Tonsillen Lacunen

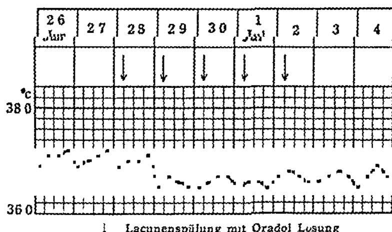
gebracht Ferner waren die Schmerzen durch Impletol Iontophorese in ca 67 % wesentlich herabgemindert

Was den Wirkungsmechanismus anbelangt lässt sich Folgendes erklären Procain blockiert wahrscheinlich synergetisch mit Koffein die aus dem Fokus über Nerven- und Gefäss-System und weiterhin bis zum Zwischenhirn zur organisierten Umstimmung führenden Impulse

Die positiven Fälle im Impletol Test wurden nach der Tonsillektomie in 85 % zur Heilung gebracht Der positive Prozentsatz bei erfolgreichen tonsillektomierten Fällen war sehr hoch und betrug 94 4 % Dieser Test stellt also ein unentbehrliches und hervorragendes Verfahren zur Herd-Diagnose der Tonsillen bei rheumatischen Erkrankungen und zugleich zur Behandlung dar Ferner kann der positive Ausfall als Indikator zur Tonsillektomie dienen Es ist auch von grossem Belang dass die Dauer bis zum Verschwinden des Schmerzens nur innerhalb von 3 Stunden bleibt und die Dauer der Schmerz-Linderung durch fortgesetzte Injektionen keinerlei verkürzt wird

2 *Spülung der Tonsillen-Lacunen* Durch Spülung der Tonsillenlacuna besonders aber der Lacuna superior werden Keim haltige Eiterpfropfe bzw Detritusmassen völlig entfernt und der Einfluss auf die daraus stammenden Fernsymptome beobachtet Die Methode wird besonders zur Diagnose der chronischen Subfebrilität verwendet Der Wirkungsmechanismus wird so erklärt dass einerseits die Resorption der Bakterien und der produzierten Toxine aber auch der Antigen haltigen Substanzen ausgeschaltet werden und andererseits nach dem gebesserten Befund von EKG auch die Beteiligung des autonomen Nervensystems geschätzt wird

Mit Hilfe eines Lacunenspülungsgerätes und zwar mit 20 cc der Oradol-Leukomycin Cathocillin Lösung usw werden die Lacunen täglich während 5—7 Tage gespült Dabei tritt die Entfieberung bei tonsillogenen Subfebrilitäten in einem sehr hohen Prozentsatz wie 95 5 % ein Anzeichen der Entfieberung kann in der Überzahl der Fälle schon nach 2 maligen Spülungen beobachtet werden (Abb 11)



↓ Lacunenspülung mit Oradol Lösung

Abb 11 Entfieberung durch Lacunen Spülungen

der Antistreptolysin Reaktion wird aber der grosse Wert besonders auf den durch Provokation herbeigeführten Titer Anstieg gelegt Ebenso bedeutungsvoll sind auch bei Rheumatismus tonsillogenen Ursprungs und Nephritis die R ntgen Provokation die Weltmann Reaktion die Remky Reaktion die Muskelfibrillenreaktion und Niederfrequenz Provokation Die Einschiebung von P³² in die Lacunen ist noch bei Nephritis und Subfebrilitäten als Fokal Test verwendbar

Übrigens ist noch die Beobachtung des Verhaltens der Allgemein und Fernsymptome nach der Provokation bzw Annulation sehr wichtig Ebenso wie diese sind auch die Messung der Leukozytenzahl am Seitenhals die Weltmann Reaktion EKG beim Herzleiden und noch die Muskelfibrillenreaktion bei Rheumatismus aber auch die Analyse der Eiweissfraktionen des Blutplasmas wichtig In Untersuchungskautelen verschiedener Reaktionen ist die Messung der Leukozytenzahl und der Blutsenkung besonders wichtig und danach die Temperatur Messung Wenn von diesen 3 Teste mindestens 2 positiv *sind so k nnen die erzielten Resultate dann diagnostisch besonders* hoch geschatzt werden Ausserdem sind noch die Schmerz Reaktion bei Rheumatismus der Harnbefund bei Nephritis und die K rpertemperatur bei Subfebrilitäten und EKG bei Herzkrankheiten naturlich wichtig Bei Rheumatismus ist der Ganslmayer Test auch bedeutsam Der Achillessehnendruck und der C R P Test sind mehr oder weniger von Bedeutung

Die Entfarbungsreaktion des Farbstoffes in Tonsillen kann gewissermassen als Merkmal gedienen Jedoch hat es den Anschein dass die Kapillarenresistenz Petechienreaktion Antistaphylolysin Reaktion Sludge Test Mester Reaktion Latex Fixationstest Penicillin Fokaltest die Provokation durch Galvanisation und Ultraschallwellen die Bestimmung der Mukoproteine und die Potenzialdifferenz Messung in der Mandel diagnostisch nur wenig wertvoll sind

*

COVER

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1962

Acta
OTO-LARYNGOLOGICA

S U P P L E M E N T U M 178

TREATMENT OF MENIÈRE'S DISEASE BY ULTRASONIC IRRADIATION

BY

ARNE SJÖBERG, JAN STAHLE,
SVEN JOHNSON and ROLAND SAHL

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

Acta

OTO-LARYNGOLOGICA

S U P P L E M E N T U M 178

TREATMENT OF MENIÈRE'S DISEASE BY ULTRASONIC IRRADIATION

Physical, Experimental and Clinical Studies

BY

ARNE SJÖBERG, JAN STAHLE,
SVEN JOHNSON and ROLAND SAHL

*From the Department of Otolaryngology (Head Prof Arne Sjöberg M D)
and the Department of Electronics Institute of Physics (Head Associate
Prof Per Arne Tove Ph D) University of Uppsala, Sweden*

CHAPTER I

HISTORICAL SURVEY

February 7, 1962 was the hundredth anniversary of the death of Prosper Meniere. It was on January 8, 1861 at the Academie de Medicine in Paris that Meniere presented his studies of the disease which was later named after him. The great importance of Meniere is that he identified the clinical conception of balance as a function of the inner ear. He died so soon after his lecture that he was never able to experience his own fame. This was delayed until 1872 when the French surgeon Duplay suggested the name Meniere's disease for that form of giddiness which with its clinical, pathologico-anatomical and therapeutic problems has engaged the otologists of the world ever since.

In this connection it should be observed that scientific otology is hardly more than 100 years old and it was typical of the times that Sir William Wilde in his famous book *Aural Surgery* of 1853 was able to say, concerning the labyrinth: "It is never the seat of surgical operation and cannot be examined during life".

In complete agreement with this conception from the 1850's is the pertinent account by Blegvad in his survey of the history of the Danish Oto-Laryngological Society how Schmiegelow, the founder of the society, as late as 1882 was recommended by a general surgeon never to touch with the knife the ear, which is so well hidden in the human body by Our Lord.

The disease is considered to be due to a vasomotor disorder with secondary development of an oedema of the labyrinth. As early as 1921 Brunner described the condition as "vasomotor otitis interna". At the present time the generally accepted theory of the aetiology is that the disease is due to periodic accumulation of fluid and/or electrolytes in the ear as the result of a disorder in vasomotor function. Basic work was carried out by Brunner (1925) and Dida Dederding & Mygind (1931, 1932, 1938). The latter workers together carried out comprehensive research on fluid metabolism in Meniere's disease and its treatment.

The theories of fluid accumulation were verified in 1938 by Hallpike & Cairns who were able to show histologically an extreme dilatation of the endolymph system. The changes corresponded to those already described by Wittmaack (1926). Later studies by Rollin (1940), Lindsay (1946) and others confirmed these observations. The condition is often called labyrinthine dropsy (Wittmaack 1926, Lindsay 1946) or endolymphatic hydrops (Cawthorne & Hewlett 1954 and others).

Up to the present time a total of only about 30 cases have been studied

histologically. However, a number of these did not show the typical dilatation (Berggren 1949, Wustrow & Borkowsky 1960). The latest report concerns the inner ears from Dida Dederding, who suffered from severe Ménière's disease. Following post mortem, Kristensen (1961) reports "beyond an enormous dilatation of the *endolymphatic system* as well in the cochlea as in the vestibulum pronounced degeneration of the organ of Corti plus the first neuron is found, whereas no definite degeneration changes can be traced in the ampullary crests, in the maculae or in the vestibular ganglion. Thus there is a very good coincidence between the histopathology of the peripheral acoustic organ and the acoustic symptoms, whereas there is a striking discrepancy between the histologically apparently normal vestibular sense organs and the formerly severe vestibular symptoms accompanied by loss of caloric reaction".

The surgical treatment of Ménière's disease was begun in the early part of the present century. It was Lake and Milligan in 1905 who, independently of each other, were the first to open the semicircular canals from the cavity of a radical operation of the non-infected labyrinth, thus diminishing the giddiness in Ménière's disease. Since that time development can be said to have followed four lines:

1. *Conservative surgical treatment* with the aim of reducing the endolymphatic pressure while partly or completely retaining the function of the inner ear. To this group belong drainage of the endolymph saccule described by Portmann (1927), decompression by fenestration (Lindsay 1949, Meurman 1951 and others) and Frenckner's operation (1952) which involves fenestration with tamponade of the semicircular canals and tympanosympathectomy.

2. *Intracranial nerve section*, where a large number of cases have been described by Dandy (1933, 1941) and Olivecrona (1943). The latter method now seems to have been given up in most hospitals in favour of the less risky procedure in the middle ear. In this connection it may be pointed out that all those cases described histologically by Hallpike & Cairns (1938) and S. Berggren (1949) died as a result of complications following nerve section.

3. *Radical surgical treatment* sacrificing the function of the inner ear. Of the older methods may be mentioned injection of alcohol into the labyrinth (Ballance 1919, Mollison 1935, 1939, Wright 1938, Erik Berggren 1939) and electrocoagulation described by Day (1946) and Herberts, Rydmark & Stahle (1954). At the present time membranous labyrinthectomy via the mastoidal approach (Cawthorne 1943, 1947) or transtympanically through the oval window (Schuknecht 1957, Cawthorne 1957) are the commonest methods and these result in freedom from giddiness and total deafness in the operated ear.

4. *Ultrasonic treatment*, which aims at selective destruction of the vestibular portion of the inner ear with preservation of hearing. A fuller account of this new method of treatment is given below.

CHAPTER II

INTRODUCTION OF ULTRASONIC THERAPY

Ultrasound has been used in medical treatment since the end of the 1930's The first important description of its effect was presented by Pohlman in 1939, who had successfully used the method for sciatica and brachial plexus neuralgia This was due mainly to the thermal effect which is produced in the tissues when the energy from ultrasound is converted into heat Ultrasonic therapy has been given for a large number of diseases of different types and favourable results seem to have been obtained in arthritis, spondylosis, sciatica, neuralgia and prostatitis, for example Attempts have also been made with malignant tumours A lucid description of the clinical results was presented by Matthes & Rech (1949) A detailed monograph on ultrasonic therapy has been written by Pohlman (1951), to which the interested reader is referred for further information

Interest in ultrasound from the medical point of view seems to have culminated in 1949 at the first international congress in Erlangen, when the collected experience of all was described Subsequently its area of therapeutic usefulness has been gradually narrowed, since the first enthusiastic reports have not fulfilled their promise However, earlier attempts to treat otosclerosis and adhesive processes (Mulvert & Voss, 1928) seem to have provided an impulse for the use of ultrasound for selective destruction of the labyrinth

From the historical point of view, it is interesting to remember that the Viennese otolaryngologists Wiethe, Wyt and Vyslonzil at the international congress in Copenhagen in 1948 gave the first report of the treatment of Meniere's disease with ultrasound In addition to five cases of Meniere's disease, they had also treated numerous cases of otosclerosis, chronic adhesive processes, post-traumatic and post-meningitic hearing impairment with favourable results The ultrasound was conveyed to the mastoid plane without operation via a rubber bag filled with water (Wiethe, 1949) In the same year Vyslonzil succeeded in experimentally abolishing vestibular function in animals with more powerful ultrasound, but first after exposing the labyrinth by operation

Research in ultrasound was also carried on at the other department of otolaryngology in Vienna where Schlander was head His collaborator Krejci published in 1952 his efficient and fundamental work. He had succeeded in selectively destroying the labyrinth in guinea pigs Krejci was able to show that the auditory function was present after irradiation by

means of Preyer's auricular reflex. In collaboration with Seidl at the Department of Physics I, Krejci built an ultrasonic transducer with a narrow tip which gave a concentrated beam of sound, and the effect of which was analysed by different methods including the Schlieren method (1952). They found that ultrasound is completely blocked by the air in the mastoid process and thus established the fundamental point that the inner ear must be dissected free by operation before irradiation. They considered earlier reports on hearing improvement after ultrasonic irradiation of the mastoid plane to be misleading and probably strongly influenced subjectively. On statistical analysis of 125 cases of hearing impairment treated with ultrasound on the mastoid plane, Krejci (1952) could find no effect on hearing.

Krejci has the honour of being the first to irradiate the labyrinth with ultrasound by the transmastoidal approach in a human being suffering from Menière's disease and thereby selectively destroy vestibular function. The operation was performed in Vienna on December 22, 1950. The dissected bony labyrinth was irradiated for 15 minutes with an intensity of 4 watts/cm². Immediately after operation the patient had a nystagmus towards the unoperated side. On the 7th day after operation a facial paralysis developed. At follow up examination 11 months after operation the patient was free from vertigo and tinnitus. Hearing was somewhat worse than before operation, the labyrinth could not be calorically stimulated and facial nerve function had returned.

Unfortunately Krejci did not have the opportunity to continue his successful research in this field.

At the meeting of the Collegium Oto-Rhino-Laryngologicum in Helsinki in 1951 the Portmann Group presented experiments in which another method was used for irradiation. In various experimental animals (rabbits, rats and guinea-pigs), they filled the external auditory canal with a fluid which served as coupling medium between the transducer and the tympanum. The ultrasound was then transmitted via the auditory ossicles to the inner ear. With a lower dosage lesions could be seen in the organ of Corti and the auditory nerve, while after a high dosage there was also destruction of the labyrinth. The team does not seem to have continued subsequently with ultrasonic research.

In the discussion following this lecture, Negus said: "If it is possible to destroy the auditory nerve cells or the hair cells in the organ of Corti, would it be possible to put the method to practical use in the treatment of Menière's disease, where an operation is employed by some surgeons? This would seem to provide a simple method."

At the international congress in Amsterdam in 1953, Arslan from Padua, presented his first report on treatment of Menière's disease with a newly constructed ultrasonic apparatus from Apparecchi Scientifici Federici in Milan. Arslan has since been the pioneer in this field and in a series of publications (Arslan 1953, 1955, 1958, 1962) he has described his results

TABLE 1 *Survey of reports of ultrasonic treatment*

Author year	No of cases	Vertigo	Hearing			Tinnitus	No of cases of facial paralysis
		Improved Cured	Improved	Unchanged	Diminished	Improved	
Krejci F 1952	1	1			1	1	1
Arslan, M 1958	256	251	30–40%		0	60–70%	
Arslan M 1961	600	95%	30–40%				
Arslan M 1962	97	83					2
Dubs R 1957		No account of results given					2
Lumsden R B 1958	22	19		15	5	7	3
Ironside W M S and Lindsay J R 1959	6	6	3	1	2	4	0
Arragno P R 1960	15	No account of results given					
Arragno P R 1962	50[a]	44	1	51	4	19	3
Angell James J et al 1960	17	14	5	4	8	10	
Angell James J 1962	103	No account of results given					5
Altmann F and Waltner J G 1961	[illegible]	63	10	53	13	25	2
Altmann F 1962	121	59	12	5	26	18	
Lindahl J W S and Robertson M S 1962	15	13	3	9	3	3	2
Cerny E et al 1962	22	No account of results given					

Total number of cases treated up to now approx 1100

[a] Included are six double sided cases making a total of 56 operations

His experience is now so widespread that at the meeting of the Collegium Oto Rhino Laryngologicum in Padua in 1960 he was able to report that 600 cases have so far been treated at his department and freedom from vertigo has been obtained in 95% and even hearing improvement in 30–40%. In a recent publication Arslan (1962) has reported 97 patients successfully treated with a modified technique

Reports of clinical results of ultrasonic treatment of Meniere's disease published as far have been set out in Table 1. This shows that the method has spread widely. A total of about 1100 cases has been published and the results seem encouraging. After Arslan the largest number of cases has been treated by Altmann & Waltner who in a careful follow up in 1961 showed freedom from vertigo in 67% and improvement in a further 17%. In a recent publication Altmann (1962) has reported the total material 121 cases in all from the Presbyterian Hospital in New York of which 62% were cured from vertigo after one radiation. Thirteen cases had to be irradiated twice and three cases three times some of them still without getting relief. The hearing was unchanged in 66%, improved in 11% and worse or lost in 21%. Tinnitus finally was unchanged in 76%. The total number of cases with facial paralysis was seven (6%)

CHAPTER III

GENERAL SURVEY OF ULTRASOUND

Definitions

In order to take the responsibility for the complicated ultrasonic treatment, it has been necessary to make a careful study of some basic physical concepts and to learn to know the mode of action of these high frequency sound waves on the body in order to be able to assess the dose of irradiation

It is well known that the human ear perceives sound waves from 16 cycles per second Tones under 16 c/s and tones over 20,000–22,000 c/s are similarly inaudible to the human ear *Frequencies which lie above the limit of hearing and are unable to produce the sensation of hearing in the human ear are known as ultrasound* The area of ultrasound is very wide and with modern methods it is possible to produce ultrasonic waves in certain materials up to 10^{10} cycles per second[1]

As is well known, many animals produce a very high frequency sound, which can also be perceived by their own sound receptors

It has been aptly said that humans can experience the silence of the forest only because our frequency threshold for hearing is so low If our ears could perceive ultrasound as well, this silence would be converted into an intense row We should, for example, be able to hear bats, who with their vocal cords produce sounds of 45,000 c/s The piping we are now able to discern from bats is only the undertones of their sounds They can orientate themselves by means of ultrasound and even catch their prey moths by an echo procedure The sound impulses are reflected from different objects in their surroundings and perceived by their ears They also avoid hitting objects in their path of flight in the dark If the eyes of the bat are experimentally covered, flight is as good as with eyes open If the ears and nose are closed they are made completely "blind" (Galambos, 1942 Griffin, 1944) This phenomenon was described as early as 1793 by Spallanzani and 1798 by Jurine without their being able to explain it Bats live on moths, which react to sound waves within frequencies of 10 to 200 kc/s The moth has a defence mechanism of dense hair which absorbs and diffuses instead of reflecting sound waves

It is astonishing to think that bats with their ingenious orientation and capturing mechanism undoubtedly can be said to have shown man the way

[1] 1 kc/s = 1000 cycles per second 1 mc/s = 1 000 000 cycles per second

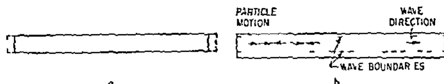


Fig 1a Total vibration of a part due to internal waves (from Benson Carlin 1960)
Fig 1b Longitudinal waves traveling through a medium (from Benson Carlin 1960)

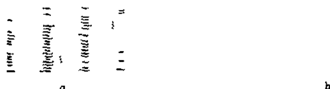


Fig 2 Particle motion in a longitudinally oscillating rod-compressional wave (a) and transversely oscillating shear wave (b) (from Lohlman 1951)

to the echo sound transmitter constructed as early as 1921 which is today used as a fathometer and for finding shoals of fish Using the fathometer it was also found that small fish could be killed by sound waves This echo principle forms the basis of all radar type systems, in which either electromagnetic or sonic waves are transmitted and reflected

We also know that with sound from a Galton pipe and similar devices, dogs can be called to place with ultrasonic frequencies of 100 000 c/s Rats hamsters and guinea pigs also perceive ultrasound at 100 000 c/s In order to demonstrate these perceptions of ultrasound use is made of Preyer's ear muscle reflexes and characteristic movements in the animal's whiskers Porpoises are said to perceive frequencies over 100 000 c/s Whales perceive and can produce sound frequencies between 20 and 30,000 c/s

The Frenchman Langevin is considered the pioneer of [illegible] ultrasonic research since he was the first to make use of what [illegible] the reciprocal piezoelectrical effect in quartz crystals and [illegible] ultrasound In 1917 Langevin was able to transmit ultrasound [illegible] 100 meters under water in the river Seine and together with [illegible] showed that strong ultrasound could kill fish This lethal [illegible] on small animals has also been shown by several other workers [illegible] van Wendt 1951) During the second world war the [illegible] used ultrasonic sirens It was also found that the power of [illegible] was high enough to ignite a piece of cotton wool, and [illegible] close were burned

As generally thought, the sound is propagated in the form of longitudinal waves Ultrasound is conveyed to the irradiated [illegible] and biological tissue in the form of longitudinal pressure waves [illegible] type shown in Fig 1 In the medium there occurs an advance [illegible] area, where

[illegible]



a b

Fig 3 *a* Particle movement due to shear waves (from Crawford, 1955)
Fig 3 *b* Total vibration of a body due to shear waves (from Crawford, 1955)

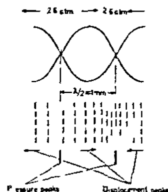


Fig 4 Displacement and pressure relationships in tissue for a sound field of 2 watt/cm² at 800 kc At the instant depicted, particle displacements have occurred as indicated by the arrows The vertical broken lines show the resulting compression and rarefaction over one wavelength Maximum particle displacement 3.1×10^{-4} cm Acceleration 0.8×10^{8} cm sec² Maximum particle velocity 158 cm/sec. Sound pressure swing ± 2.6 atmospheres (From Pohlman 1951)

every microscopic particle is exposed to alternating positive pressure and negative traction forces, as shown by the arrows The whole wave movement advances in the direction of the right arrow

Cyclic rarifactions and compressions occur In some materials, especially solids, such as bone and metals, transverse particle movements must be *reckoned with, which result in what are known as shear waves and at* times also nonpenetrating surface waves (Figs 2 and 3)

In the ultrasonic field acceleration forces occur which reach their maximum when the pressure forces are also maximum, i e at the peaks of a number of the cycles (Fig 4 after Pohlman, 1951) The active components *are thus these positive pressure and negative traction forces together with* acceleration forces

The ultrasonic power is given in *watts*, by which is meant the rate of energy coming from the active area (the treatment area) of the transducer The power radiated is also determined by the size of the area of the transducer *The relationship between power and area is expressed as in*tensity (watts cm²) The total energy output can be expressed in *joules* (watts × time in seconds)

Finally the ultrasonic frequency is important, for medical treatment being between 175,000 and 3,000,000 cycles/second In the treatment of Meniere's disease about 1,000,000 cycles/second (1mc/s) are used In a recent report, however, Angell James *et al* (1963) have used 3 mc/s

Biological Mode of Action

The biological mode of action of ultrasound is complicated and not completely clear In general it seems to be based on three factors (1) mechanical, (2) thermal, and (3) chemical

The high frequency ultrasonic waves penetrating the body are reflected, absorbed and can give rise to standing waves when they pass through different media represented by various tissues, such as skin, connective tissue, bone, blood vessels, muscle, etc Because of this, it is not possible to give exactly the ultrasonic energy acting in the tissue In other words, the irradiation dose cannot be given exactly as the difference between input and output, as with an ionized irradiation–X-ray treatment *The effect on the tissue can only be given biologically (coagulation, oedema, paresis, nystagmus, etc)*

1 *Mechanical effect* Mechanical forces of considerable size develop during the passage of ultrasound through tissue Elementary particles in the exposed medium can be accelerated to 100,000 times the force of gravity by ultrasound with an intensity of 5 watts/cm² at a frequency of 800,000 cycles/second (Baldes, 1958) Expressed simply, the effect of ultrasound on the tissue can be described as a shaking movement—a "micromassage" This also results in the production of bubbles and gives the impression of boiling without any appreciable temperature rise Dispersion can be so powerful that real fog formation occurs This effect of ultrasound is used for mechanical cleaning of different types of instruments both in medicine and in industry Recently this effect has been used for aerosol treatments

Ultrasound also has a sterilizing effect Uni- and multicellular organisms such as bacteria, frogs and small fish can be killed by irradiation Electron microscopy has shown that virus are broken down and bacterial membranes ruptured In a suspension of erythrocytes the cells are first deformed then become pale and finally burst with the development of haemolysis when irradiated Destruction of red cells has been used as a test of the biological action of ultrasound (Dognon & Biancani, 1937) Leucocytes are also destroyed

One expression of the mechanical effect is called *cavitation* This phenomenon occurs because, under certain conditions, such a low pressure may occur in the wave troughs that fluid particles are broken up and a hollow space containing a gas phase is formed Cavitation is especially marked in boundary areas between solids and liquids The haemolysing effect of ultrasound is thought to be due to cavitation Even small eggs of Arbacia

(diameter 75 micra), which are approximately the size of blood cells burst apart through ultrasound as shown by Harvey & Loomis (1931) with microfilming

2 *Thermal effect* The thermal effect of ultrasound depends on an absorption of energy in tissue The amount of transmitted sound energy which is converted to heat is determined by the absorption coefficient of the tissue Bone has a very high coefficient and therefore becomes very hot An essential part of the destructive effect of ultrasound when used in the treatment of Meniere's disease is possibly caused by heat

This heat has been previously used with a certain amount of success in human medicine in the same way as in short wave treatment (cf p 9) However ultrasound as a source of heat has been used less and less, since almost the same effect is obtained with short wave therapy

3 *Chemical effect* The chemical effect is complicated and variable A number of workers consider cavitation to be the requirement for the development of chemical reactions Cavitation leads to increased ionization of water, so that free radicals (OH) appear, which are very active and have an oxidizing effect (Pohlman 1951) For example, ultrasound can release iodine from potassium iodide (Graber & Prudhomme 1949), and aromatic groups all undergo great changes The observation of Schmid (1949), that ultrasound can break down macromolecules is also of importance Finally it is known that many chemical reactions can be started or accelerated ultrasonically

The effect of ultrasound on bone and nerve tissue is of the greatest importance from the otological point of view, and a more thorough account will now be given

The Effect of Ultrasound on Bone

When ultrasonic energy penetrates bone a large portion is changed to heat because of the very high absorption coefficient The term half-intensity distance is the distance which ultrasound traverses in a medium before half of the original intensity ($^3/_4$ of the energy) is lost This is very short for bone about 0 5 mm while for water it is about 15 m (Angell James *et al* 1960)

The conversion of ultrasonic energy to heat can be illustrated by Schlieren photography (fol p 34) When ultrasound comes into contact with a slice of bone placed just in front of the tip of the transducer in a Schlieren system a zone of intense light appears around it This is mainly an indication of the heat development in the bone (Fig 17) It is known from several experimental investigations that ultrasound even in comparatively small doses can injure both growing and adult bone Buchtala (1948) and Barth & Bulow 1949) irradiated the extremities in puppies, which developed inhibition of growth and also more severe degenerative changes ending in

spontaneous fractures 3–6 weeks after irradiation The latter authors used an intensity of 3 25 watts/cm² for 15 minutes Similar results were presented by Arden *et al* (1957), who used 2 watts/cm² for 5 minutes with a transducer whose 'area of soundhead' was 10 cm² Histologically, the injuries appear mainly in the boundary layer between different media (soft tissues, periosteum, bone) where absorption of heat is maximal (Majno, 1949, Bender *et al*, 1954)

A higher ultrasonic energy up to 50 watts/cm², and in most experiments a constant irradiation of the dog's femur for 30 minutes, has been utilized by Janes *et al* (1962) at the Mayo Clinic This dosage produced a total necrosis, including destruction of the blood supply of an irradiated region of the bone Proximal and distal to the area of necrosis developed periosteal and endosteal formation of new bone with marked increase of the periosteal and endosteal vessels

The Effect of Ultrasound on Nerve Tissue

Nerve tissue is the most sensitive of all human tissue to ultrasound White matter is considerably more sensitive than grey matter (Barnard *et al*, 1955, 1956, Fry *et al*, 1955, Ballantine *et al*, 1956, Åstrom *et al*, 1961) This means that fibre tracts can be interrupted without disturbance to neighbouring or surrounding grey matter (Barnard *et al* 1956) The order of sensitivity in white matter is as follows myelin sheaths, axon cylinders, glia with blood vessels the least sensitive It has been possible to produce small, well limited lesions in the central nervous system using stereotaxic methods and focussed ultrasound (Fry and co workers, 1955, Fry, 1958, etc) Afterwards they could follow the degenerative changes histologically in the nervous pathways and obtain important information and knowledge They used ultrasound with a frequency of about 1 mc/s produced in four separate quartz crystals and focussed with four lenses All these have a common focal point in which the intensity of irradiation can be as much as 400 watts/cm² Small selective lesions were produced in experimental animals after irradiation with 200–400 watts/cm² for 4 seconds (Fry *et al*, 1955)

Åstrom *et al* (1961), using a single quartz crystal have focussed the sonic beam on the subcortical region in cats and produced pan necrosis within this region The cats were irradiated after craniotomy at a frequency of 2 7 mc/s The peak intensity at the focus was 1700 watts/cm² and dosages were graded by varying the exposure time within the range of 0 25 0 4 seconds At the same laboratories Lele (1962) and Basuri & Lele (1962) have shown that a preselected (grey or white) target in any location within the cranium can be destroyed accurately by means of a stereotactic procedure

If the peripheral nerves are irradiated with varying times and intensities

different degrees of functional impairment are obtained (Madsen & Gersten, 1961). *Young & Henneman (1961) have shown that the largest fibres in* nerve trunks are the least sensitive to ultrasound

A number of authors consider that impairment of function is mainly caused by intraneural heating (Herrick, 1952, Baldes *et al*, 1958). Andersson *et al.* (1951) showed that an action potential produced experimentally diminishes in amplitude after both ultrasound and heating If intraneural heating exceeds 45°C, total and irreversible block usually occurs From the otological point of view, the close relationship of the facial nerve to the labyrinth is important Fry (1953) and Barnard and co-workers (1955) were of the opinion that ultrasound also has a nonthermal effect on nerves This was supported by experiments on cooled frogs in which measurements with thermocouples showed that the temperature never exceeded 25°C at the site of operation In spite of this the animals showed degenerative injuries Mechanical (Ballantine *et al*, 1956) and chemical factors probably contribute to the lesion It is well known that in experiments *in vitro* ultrasound affects enzymatic processes, changes pH and has colloid chemical effects

Histological investigations after ultrasonic irradiation of the spinal cord showed that hyperaemia develops immediately, followed by oedema after six hours (Peters, 1949), and commencing of degenerative changes in nerve and ganglion cells after twelve hours These changes increased during the following days. It is presumed that the initial oedema produced arrest of the circulation with ischaemia and degeneration as the result (Pohlman, 1951).

The sensitivity of peripheral nerves to ultrasound as well as heating makes cooling of the apparatus necessary If the tip of the transducer is hot, as in the Italian apparatus, this heat is also transferred superficially to the labyrinth (Angell James *et al*, 1960, 1961; Altmann & Waltner, 1961) In this way alone there is a caloric stimulation of the labyrinth and the risks of thermal damage to the facial nerve increase At the international symposium in Padua in 1962, Altmann described the effect of "excessive overheating" resulting in postoperative necrosis of the thinned out wall of the lateral semicircular canal, facial paralysis occurred in seven out of 121 irradiated cases Using a thermocouple we have been able to *establish that even with an output power as high as 2 watts, the tip of our Swedish soundhead is heated to less than 1or 2°C above the temperature of the circulating water (about 35°C)*

Apart from the treatment of Menière's disease, high frequency ultrasound is used clinically to a *limited extent both therapeutically and* diagnostically Therapeutically, the destructive effect of ultrasound is used in neurosurgery Prefrontal lobotomy was carried out by Lindström (1954) using unfocussed ultrasound (7 watts/cm², 1 mc/s, irradiation time 10 min) with satisfactory clinical effect However, in order to allow a sufficient quantity of ultrasound into the brain, two openings must first be made

in the skull which would otherwise itself absorb most of the energy Postmortem studies from the Lindstrom material showed small histological changes localized mainly to the white matter

Using a stereotaxic instrument and focussed ultrasound Leksell (1957) carried out pallidotomy with good results However he asserted that stereotactic irradiation treatment has hardly begun yet Fry (1958 1962) and Myers and co workers (1959) have successfully treated some 50 Parkinson type patients Attempts have also been made to destroy the hypophysis in patients with mammary cancer in advanced stages (Fry 1962 Fry & Fry 1962)

The first to use ultrasound diagnostically was probably Dussik (1942) who examined the skull with a transmission method The paths of the ultrasonic waves through the skull were recorded with a cathode ray oscilloscope with which a complicated picture known as a hyperphonogram was obtained A further development of this line can be said to be Leksell s echo encephalography (1955) In this case ultrasound is transmitted in the temporal region where the bone of the skull is thinnest The ultrasound is partly reflected by the falx cerebri so that what is called a midbrain echo occurs A barium titanate crystal is alternately transmitter and receiver and the echo is recorded on a cathode ray oscilloscope The investigation indicates deviations of the middle brain line caused by extensive processes such as tumours and intracranial haemorrhages (Leksell 1955 Lithander 1961 Jeppsen 1961)

Ultrasoundcardiography as described by Edler (1961) follows similar principles Echo signals can be obtained from various parts of the human heart *in vivo* The movements of the heart can be recorded continuously and in ultrasoundcardiogram is obtained Using this method the movements of the heart valves have been mapped out in healthy and sick people

ULTRASONIC GENERATORS IN OTOLOGY

Other Equipment

Ultrasonic generators intended for percutaneous treatment of rheumatic diseases neuralgias etc generally have too low an intensity for them to be useful in the treatment of Menière s disease A number of irradiators capable of destroying the labyrinth in man are described

1 The first apparatus intended for destruction of the inner ear was constructed by *Fa F Reiner in Vienna and described by Krejci in 1952 In order to direct the beam of rays towards the dissected labyrinth* the ultrasonic transducer was provided with a small tip of glass filled with water By this means *a relatively well concentrated beam* was obtained with a diameter of 4 mm *In the only* apparatus described by Krejci the intensity was 4 watts/cm² and the irradiation time 15 minutes with a *frequency of 800 kc/s*

2 The second apparatus was constructed by Apparecchi Scientifici Federici in Milan following instructions by Arslan (1953) It consists of an oscillator assembly and a treatment head known as the transducer The ultrasound is produced by a quartz piezoelectric disc 4 1 cm in diameter lying inside the transducer the tip of which has a diameter of 5 mm The treatment area is thus 0 2 cm²

According to Arslan treatment should begin with an initial intensity of 7 8 watts/cm which can be increased up to a maximum of 15 watts/cm² The character of the nystagmus is decisive for the duration of irradiation which in principle extends till the moment when the nystagmus clearly beats away from the irradiated side known as paralytic nystagmus (cf p 54) The Italian apparatus and the same irradiation technique have been used up to now in a number of hospitals (Dubs 1957 Lumsden 1958 Lindsay & Ironside 1959 Altmann & Waltner 1960 Aragno 1960 etc

3 Modifications of the Federici Generator Angell James and co workers in Bristol have modified and improved the Italian apparatus Cooling takes place by increased flow rate of the water oil suspension controlled by a new flow indicator Two instruments for measurement of ultrasound have also been constructed by this team an absorption calorimeter and a new type of intensity meter

The Angell James group has shown by the Schlieren method that their apparatus gives a well concentrated beam of rays Their studies of temperature in experimental operation cavities are important They measured the temperature at the tip of the transducer for varying intensities Without cooling, it rises to 90°C, which must involve great risks for the facial nerve The temperature in the contact area between the transducer and the bone can be reduced to 44°C only when the operation cavity is cooled with 200 ml/min circulating water at 39°C In clinical operations they use continuous irrigation with saline at 37°C, the method also used by Altmann & Waltner (1961)

The Bristol group has recently (Bullen *et al*, 1963) constructed a new apparatus, which differs in principle from previous irradiation equipment The treatment head consists of an air-sleeved distal crystal having a tip diameter of 3 mm, ultrasound being generated by means of a lead-zirconate-titanate piezoelectric element of 5 mm diameter, recessed in a conical casing close to the tip In spite of continuous saline irrigation during the operation, the tip temperature reaches 47–52°C at intensities of 25–40 watts/cm² Forty-seven patients have been treated with this new equipment, of which four received facial paralysis

Gordon (1960) has constructed a cone-shaped treatment head in which a lead-zirconate crystal is cemented to a brass exponential velocity transformer, ending in a cylindrical interchangeable application tip The transducer is cooled by means of circulating water, which is allowed to flow out through the tip into the operation cavity The available output is 8 watts/cm²

The Italian apparatus even in its modified form, has a number of disadvantages, many of which have already been pointed out by Angell James and co workers (1960, 1963) A number of clear disadvantages may be mentioned

1 The treatment head (= the transducer) is large and clumsy and therefore difficult to handle

2 Ultrasound is produced by means of a quartz crystal and requires a very high alternating electric potential to be brought into vibration This makes a thick electrically well insulated cable necessary, increasing the weight of the treatment head considerably

3 As pointed out above, the tip of the transducer becomes excessively warm during irradiation (up to 90°C) A large amount of heat in the contact area is undesirable and carries a risk of damage to the facial nerve (Altmann & Waltner, 1961, Altmann, 1962)

4 The transducer as a whole cannot be sterilized

5 The tip of the transducer has too large a diameter (5 0 mm), which impedes exact application In addition the tip must be shielded to reduce undesirable lateral irradiation which can damage the facial nerve, dura and brain

The New Swedish Ultrasonic Generator

With these facts as a background, *the new Swedish ultrasonic apparatus, Ultrapoint*, has been produced in Uppsala with intimate cooperation between otologists and physicists. The equipment consists of two main parts (Fig. 5).

1. *The generator*, which includes a radio frequency oscillator, r.f. power amplifier and power supply.
2. *The treatment* head with its cooling system.

Two ultrasonic generators were made, the first of which can be said to be conventional in type. This was built by the Scientific Research Council's Electronics Division at the Department of Physics of Uppsala University and was used in all the preliminary experiments as well as in the first 20 operations. In order to be able to carry on experimental research and clinical treatment at the same time we decided to build another unit. In the latter, built by one of us (Johnson), both the oscillator amplifier part and the power supply have been considerably simplified and the unit has been given a modern design. The latter new construction is illustrated diagramatically in Figs. 6 and 7.

This new apparatus consists of an electron coupled r.f. oscillator power amplifier. Instead of varying the output of the power amplifier by means of an adjustable plate potential, as is generally done, it is operated at a fixed, constant level. A matched variable attenuator delivers a specific portion of the maximum power to the treatment head. Calibration is accomplished by measuring the power radiated from the treatment tip with the power level adjustment set at 100. The radiated power is measured by means of a calorimeter or a new intensimeter to be described below. After this, the output power may be adjusted in 10% steps from 100% to zero.

By virtue of the fact that the bandwidth of the water loaded transducer is relatively broad, this is tuned by means of a separate fixed inductance L_6 (Fig. 7).

One feature of the oscillator portion of the unit is the use of a high stability circuit generally known as a Clapp or series tuned Colpitts oscillator. Since the anode tuning, determined by C_{11}, is relatively insensitive, readjustment during an operation is not required, once properly tuned. This electrical stability, together with the broad response (compared to a quartz crystal) of the lead zirconate transducer, makes the entire system extremely uncritical and stable in use. Any temperature sensitivity of the transducer (variation of resonance with temperature) is circumvented by the cooling system, which maintains the entire transducer and tip assembly at a constant temperature.

The treatment head (transducer probe) is the essential new construc-

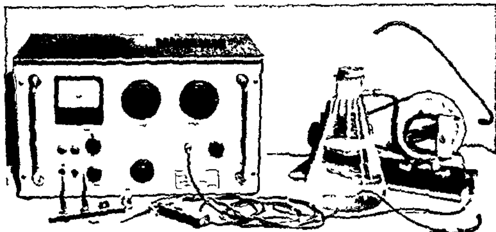


Fig. 5 The Swedish ultrasonic generator *Ultrapoint*. To the right is seen the pump and container for the cooling water.

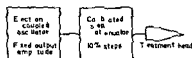


Fig. 6 Block diagram of the main components in the Uppsala ultrasonic irradiation apparatus.

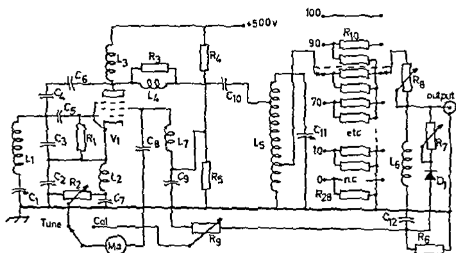


Fig. 7 Basic schematic of oscillator and attenuator portions of the ultrasonic generator

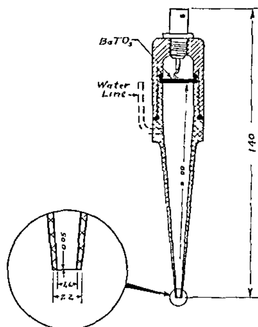


Fig 8 Diagrammatic view of the new Swedish ultrasonic treatment head The ultra sound is generated by means of a barium titanate or lead zirconate titanate disc, and is focussed just inside or in the tip of the conical head Cooling is effected by means of circulating distilled water inside the cone

tion, in which the disadvantages of earlier equipment have been eliminated A more complete description follows (Figs 8 and 9)

1 Its length, including BNC contact, is about 140 mm and its weight about 100 g, depending on the shape and size of the tip Its greatest diameter is 25 mm and it can be held like a pen A slip on handle may be applied, further facilitating treatment (Fig 10) The tip of the treatment head, which is hollow, is closed in front by a very thin metal membrane

2 The ultrasound is produced by a concave disc of barium titanate or lead zirconate This requires an electric potential of only a couple of hundred volts for maximal clinical power On the other hand, a quartz crystal requires several thousand volts for the same result Because of this, a thin and flexible cable can be used

3 The treatment tip is continuously irrigated with distilled water, driven by a special circulating pump (Fig 5) The water has the double function of conducting the ultrasound from the crystal to the tip of the transducer and of cooling the unit The entrance and exit for the water are clearly shown in Fig 10 as two bent tubes With the range of power which we have used clinically so far, the temperature of the tip (at the very end) exceeds that of the circulating water (approx 35°C) by only 1–2°C

4 The whole treatment head, including the cable and the cold water tubing can be sterilized by boiling or autoclaving For this reason they do not need to be covered with sterile drapes during operation

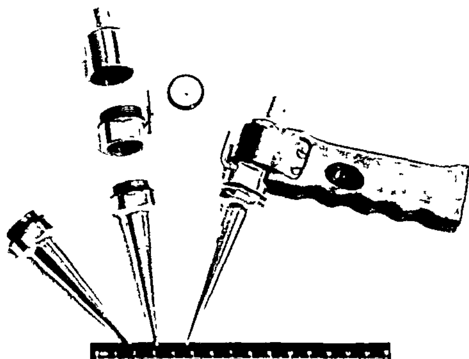


Fig 9 The ultrasonic transducer—to the left disassembled to show its components to the right equipped with handle A later type of handle is shown in Fig 10

5 The actual tip of the treatment head, which is detachable and interchangeable, has been made in several sizes, the diameter of the tip varying from somewhat under 1 5 mm to about 2 6 mm Obliquely bevelled tips have also been produced to facilitate application in cases with difficult anatomy (Fig 11) The smaller tips have the advantage that they can be brought into contact with the semicircular canals more exactly than the wider ones With our tips, the operator can see the application point in relation to the rest of the labyrinth the whole time This makes the irradiation more exact The tips having the smaller diameters are very well suited for animal experiments

There is a difference in principle between the Italian ultrasonic transducer (Federici) and ours In the Italian apparatus the quartz crystal is in liquid contact with an aluminium cone, which is drawn out to a tip 40 mm long with a final diameter of 5 mm Ultrasonic energy radiates from the flat end of this 5 mm diameter tip, which must be regarded as the source of ultrasound as far as the liquid and tissue in contact with the tip are concerned Effectively, the radiating surface in our new apparatus

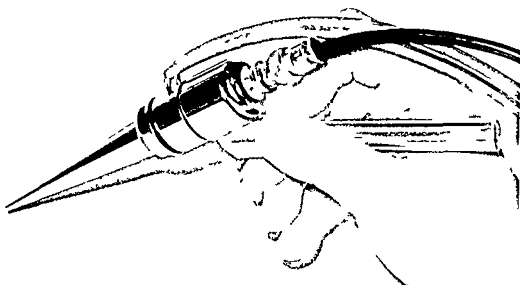


Fig. 10. The transducer equipped with handle. On the top can be seen the hoses for the circulating water.

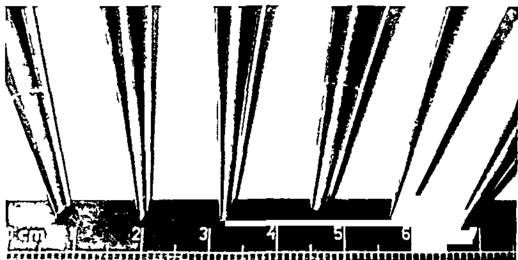


Fig. 11. Application tips for the transducer. The tip diameters range between 1.5 and 2.6 mm. Some are bevelled.

is that of the actual transducer itself, nearly 20 mm in diameter. Virtually all of this energy is brought to a focus at the apex of the conical treatment tip. As a result a minimum amount of energy will pass through the side wall of the cone and though there is spreading due to diffraction effects at the membrane covered opening, this is reduced considerably by the focussing of the transducer.

A great advantage resulting from the small diameter of the probe, together with the fact that no heating problem calling for outside cooling exists, is the possibility of locating the tip precisely on the portion of the labyrinth one wishes to irradiate, the only liquid necessary around the tip being a very small drop between the flat membrane at the end and the bone itself.

The quantity of energy emitted by the tip of the transducer has been measured calorimetrically in accordance with the manner of Angell James and co-workers (1960) and may be checked during operation by an ultrasonic monitor. Tip intensities approaching 200 watts/cm² have been achieved with the new Swedish apparatus. Such an intensity was considered desirable especially for some animal experiments.

A comparison of the intensities given by Arslan, the Angell James group and others as well as those we have used, can be bewildering. However, it must be remembered that the frontal area (= treatment area) is considerably smaller in our apparatus than in the Italian. Depending on which size of treatment tip we choose to employ, the frontal area varies between about 1.7 mm² and 5 mm² (diameter about 1.5–2.6 mm). In the Italian apparatus the treatment area is 20 mm². The ratios between ours and the Italian are $^1/_{12}$ and $^1/_4$ respectively. The power yielding an intensity of 10 watts/cm² in the Federici apparatus, which is a common dosage in clinical work, results in our apparatus in an intensity of 120 watts/cm² and 40 watts/cm² respectively, depending on which tip we use. *As a result of this and to avoid misunderstanding, we have chosen to express the total power emitted from our apparatus in number of watts and not, as with Arslan and others, in watts/cm².*

To summarize briefly, our new apparatus is constructed in the following way. High frequency alternating current is transferred from the oscillator to the transducer, in which a small concave barium titanate or lead zirconate disc converts the electrical energy into ultrasonic waves. The beam of rays is well concentrated with the focal point just inside or in the flat top of the conical treatment head. Cooling is effected by means of circulating distilled water. The conical tips on the transducer are interchangeable and vary in diameter from 1.5 mm to 2.6 mm.

CHAPTER V

PHYSICAL EXPERIMENTS WITH THE SWEDISH ULTRASONIC GENERATOR

Measurements of the Temperature at the Tip of the Transducer

The temperature at the tip of the ultrasonic transducer was studied in a series of experiments A small copper plate, connected to a thermocouple, was applied to different places around the tip of the transducer The plate and the thermocouple were enclosed in the bottom of a conically bored plastic container, the inner dimensions of which corresponded exactly to the shape of the ultrasonic transducer A similar thermocouple immersed in ice water at 0°C was used as reference electrode The changes in temperature were read off on a galvanometer which had been calibrated against known temperatures

In these experiments rather large differences in temperature were obtained varying with the power emitted from the ultrasonic transmitter The reason for this was that when the ultrasound encountered the copper plate, the ultrasonic energy was converted to heat within it For this reason it was impossible to measure exactly the increase in heat on the tip of the ultrasonic transducer On the other hand we succeeded in measuring the increase in temperature on the side of the transducer, just beside the tip Here the rise in temperature was about 1 2°C with an emitted power of 2 watts The same value may be expected even at the tip of the transducer (the area of treatment)

The Measurement of Ultrasonic Output

The quantity of energy emitted from the tip of the ultrasonic transducer was measured calorimetrically in agreement with the suggestion of Angell James *et al* (1960) Care is taken to measure only that energy leaving the very tip of the transducer cone so as to avoid any error from sidewall radiation Thus, the tip is not immersed into the liquid of the calorimeter, but is held just in contact with the liquid surface

Maximum ultrasonic power output of the new apparatus is to a degree a function of the tip diameter *In general however, for tip diameters of*

2 6 mm down to and including 1 5 mm, the limit lies somewhat over 3 watts By means of the "K-factor" control, which adjusts the loading on the attenuator to a constant value, the output has generally been adjusted to an even 2 watts The meter can be set to give a reading of full scale by means of R9 (Fig 7) at 100 on the "power level' scale, when set in the "cal" or calibrate position Once this has been accomplished, power levels may be chosen in 10% steps from 100% down to 0 If any change occurs in the transducer, or if a special tip is employed, its output may be measured at the 100% position, and the power available at any level is easily seen to be simply that percentage of the maximum

An ultrasonic piezoelectric monitor, in general like that described by the Angell James group, has been constructed This apparatus makes possible a quick check of the amount of ultrasound emitted during the actual operation We have however, also constructed a new, simple unit described below

Intensimeter

A new device for measuring the ultrasonic output of the treatment head has been designed Since it is new in principle of operation, it has been called simply an "Intensimeter' By measuring the amount of circulation *or streaming in a liquid into which the tip is immersed, it is possible to* obtain a measure of the actual acoustic power radiated

The device constructed (Fig 12) consists principally of a pyrex glass tube formed into a 'd' This 'd is open at the top, the loop re-entering the upper part of the tube approximately 5 cm from the top The short side of the loop of the 'd' is slightly conical narrow (3 mm dia) at the bottom In this cone is placed a small, about 4 mm diameter spherical bead The tube is now filled with a liquid, distilled water is generally used When the treatment head tip is set down into this and turned on the liquid will be made to circulate within the loop of the 'd" and if sufficiently strong will cause the bead to rise in the conical tube The lifting force on the bead is a function of the velocity of liquid flowing past it Once this reaches a point at which this force exceeds the weight (gravitational pull downward) the bead will lift By making the tube conical, the velocity of the liquid will vary, being greatest at the narrowest, bottom portion, falling off as the cone widens Thus, for a given intensity of ultrasound the bead will rise to only a certain point *and remain there as long* as the ultrasonic power remains unchanged Through the use of a calorimeter it is then possible to calibrate the bead position in terms of radiated ultrasonic power This has been done for the model shown (Fig 12) which covers a range of 1 5 to 3 0 watts

In use the generator is set at 100% output the tip inserted into the intensimeter and power turned on Tuning for maximum output is easily

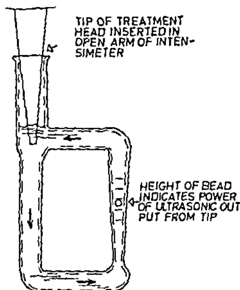


Fig 12 The Intensimeter By measuring the amount of streaming produced by the ultrasonic output of the treatment head the Intensimeter provides a very quick and reliable method for the determination of power output

accomplished by observing the position of the bead The power level thus indicated is the maximum (100%) output of the apparatus for the particular transducer or tip employed

By varying the taper of the conical arm of the intensimeter, as well as the weight of the bead, any range of power may be measured Thus a low power intensimeter can easily be made to cover the range, say from 0 1 to 1 watt, or any other so desired A power check takes only a matter of a few seconds

Studies of Ultrasound by means of a Reflection Schlieren Optical System

The Schlieren method for investigating small variations in the optical properties of a transparent medium is employed regularly in, for example, ballistics research, where shock waves generated in air by the passage of a bullet or other missile, may be seen and photographed Similarly, shock waves generated in wind tunnels on airfoil sections or rocket models are studied by this method Glass quartz and other materials are often quality tested by Schlieren optics Innumerable other applications could be listed having the same principle of operation, although the specific instance may call for considerable variations in method

The one essential point in common is that of letting light diverging from

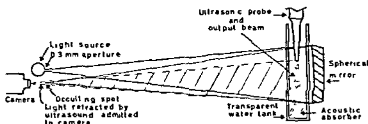


Fig 13 Diagrammatic view of the Schlieren system used in our investigations All of our pictures of ultrasonic beams are photographed using this method

a point or slit source pass through the medium to be studied, then refocussing to an image This image, of the point or slit light source, is allowed to fall on an occulting spot or wire, preventing the light from going further If the occulting spot or wire is just large enough to block the light in the system, but no larger, any variations in the path, for example in the study medium, will cause some light to pass by the occulting point If, further, one either views or places a camera behind this point, one may then see or photograph the regions of the medium having caused the light to diverge from the basic simple system In practice, the light from the source is often collimated, and as a bundle of parallel light rays is allowed to pass through the medium or region under examination, then refocussed after this point Either a transmission system employing lenses or a reflection system consisting of one or more mirrors may be used

The single spherical mirror system which we have employed gives essentially the same results as obtained from the two mirror, Toepler system as long as the focal length of the mirror employed is long relative to the area viewed This method has the added advantage of a greater sensitivity, since the light passes through the same area twice, and suffers twice the disturbance The disturbances we make use of to show the ultrasonic radiation pattern are the alternate compressions and rarefactions in a liquid (*water*) *produced by the ultrasonic waves emitted by the transducer* These result in changes in the index of refraction of the liquid, thus causing light to be deflected out of the basic Schlieren optical path and allow it to bypass the occulting spot and into the camera lens The camera, focussed on the plane containing the probe tip, will then record the regions having produced the light deflection, in other words, the ultrasonic radiation pattern Our optical device is schematically illustrated in Fig 13

A number of important factors must be taken into consideration when viewing and attempting to interpret a Schlieren photograph The first of these, in connection with the study of the radiation from the ultrasonic treatment head with which we are concerned, has to do with the geometrical shape of the ultrasonic field going out from the tip It is clear

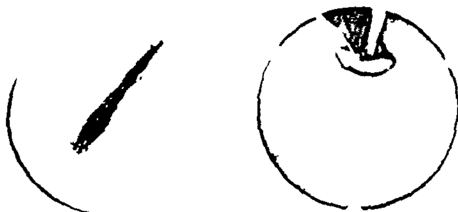
dependence, so that the entire phenomenon of seeing a Schlieren image of the ultrasonic radiation from a small source, such as that of our treatment tip, *can at best be taken only as a qualitative guide to the general sound energy distribution in its field*

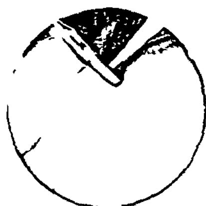
It is possible, however, to draw some valuable conclusions from Schlieren photographs, such as the series shown in Figs. 15 A–D Here is illustrated the non-linear dependence of light output from the Schlieren system versus ultrasonic power One can say with certainty that the intensity of the ultrasonic field found in the side lobes will be of a considerably lower value than in the central cone This may also be inferred from the series in Fig 15 If we further consider the light paths through the various portions of the sound field which we observe, it is easy to see that the central cone affects the light over only a short traverse, while the outer regions (the rings in the target analog) will affect the light for a greater distance, when travelling tangentially to the midregions of each ring There is thus a good chance that the combined effects of sound intensity plus distance traversed will yield the same light output from the Schlieren system, so that both central ray and side lobes will appear equally bright when observed or photographed

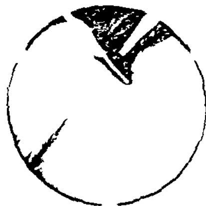
It is to be pointed out that the experiment illustrated in Figs 15 A–D was made in water, resulting in a clear side lobe radiation If the sides of the transducer cone is surrounded by air, no side-ward radiation can exist In clinical use the tip is always kept clear of any liquid, except at the very end, and contact with the sides of the operating cavity is avoided

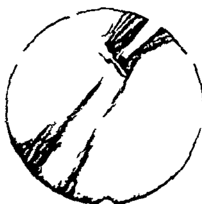
In order to obtain at least a rough idea of how much of the applied ultrasound is absorbed in bone, this problem has been studied in the same manner as done by Angell James *et al* Flat slices of bone were placed in the Schlieren system water tank, a few mm in front of the transducer tip and the relative intensity of the sound field before and after traversing the bone was noted The pieces of bone were taken from the femoral diaphysis, sawn and filed down until thicknesses of 0 2 mm–4 mm were obtained In these experiments the ultrasonic power output was 2–2 5 watts and the diameter at the tip of the transducer 2 6 mm *With a thickness of bone of 4 mm, no ultrasound is seen to penetrate. All radiation is either absorbed or reflected* (Fig 17) *The light zone around the piece of bone is caused by heat, due to absorbed ultrasound (cf p 16). A piece of bone of 3 mm thickness transmits a minimal quantity of ultrasonic rays* (Fig 18) *With thinner layers of bone of 1 0–0 5 mm there is a clear penetration but there is still considerable reflection and absorption* (Figs 19 and 20)

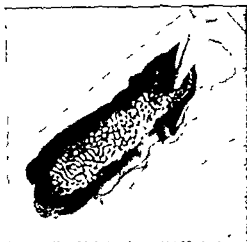
Our experiments corroborate that ultrasound is largely reflected and absorbed by bone Only with a thickness of bone of 1 mm or less does a clear penetration occur, which is also in agreement with our clinical observations Our findings that a clear penetration occurs of bone slices 1 mm thick differ to some extent from the results obtained by the Angell James


16 17


18


19


20


21

Figs. 16–20. Schlieren photographs illustrating the ultrasonic beam and its penetration characteristics through bone (femoral cortex) of different thicknesses. A strong reflection and absorption takes place in the entire bone slice (Figs. 17–20) which causes the illuminated zone around the bone.

4 mm thick bone is not penetrated (Fig. 17).

Bone slices 3, 1 and 0.5 mm thick allow penetration of ultrasound in increased amounts (Figs. 18, 19 and 20).

Fig. 21. Standing waves in blood, brought about by ultrasound with a power of 0.5 watt (standing wave test[7]).



Fig 22 Standing waves in powder produced by the reflection of soundwaves against a concave mirror (from Straubel, 1931)

group in similar experiments (1960) With Schlieren photography, they have shown that due to interference of directly transmitted waves with waves reflected from both faces of the bone lamina, a 1/4 wavelength lamella (1 mm thick) gives almost no transmission of ultrasound, whereas there is good transmission with thickness of 1/4, 1/2 and even 2 mm. However, in bone pieces 2, 3, 4 mm in thickness a very large quantity of energy is lost as the result of absorption (cf Figs 16–20)

As a result of our experiments we thin out the bony wall of the lateral canal with a diamond drill until we just catch a glimpse of the grey line, a technique used also by the Angell James group and Altmann & Waltner (1961) Furthermore as Arslan (1962), we make a groove behind the lateral canal over the vestibule leaving only a thin layer of enchondral bone *The results of the transmission experiments also give us a certain safety margin They allow us to assume, on good grounds, that no ultrasound with destructive energy is transmitted through the innermost parts of the labyrinthine capsule and then intracranially*



Fig. 23. Standing wave patterns produced in fine powder (reproduced from Crawford 1955).

Functional Tests with Standing Waves

A method for visualizing standing waves by means of particles of dust was described by Kundt in 1866. Similar methods are also of use with ultrasonic waves, as long as the wavelength is not less than about 1 mm (Bergmann 1949). For dust, lycopodium powder is recommended. This collects in the nodes of the waves in narrow strands. The distance between these strands is ½ the wavelength, so that the wave pattern permits a coarse assessment of the wavelength. A condition for the appearance of standing waves is that the ultrasonic waves are reflected by a boundary. Fig. 22 shows a beautiful standing wave formation in lycopodium powder, where the source of ultrasound consists of a swinging quartz rod and the reflector of a concave mirror (Straubel 1931).

In addition to lycopodium powder, fine elder pith, kieselguhr and fine quartz sand have been used. A further example is given in Fig. 23 (from Crawford), which demonstrates the presence of ultrasound in an absolutely convincing way. Standing waves can also be elicited in fluid media, e.g. slow flowing oil, coloured liquid paraffin, water with fine charcoal or



Fig. 24. Sand sculpture. Picture taken by the Swedish nature photographer Pål Nils Nilsson.

pumice powder as in suspensions and emulsions (Marinesco 1932). A striking resemblance to this phenomenon can be found in nature on a sandy beach after a storm where the waves of the sea remain, which is shown very well by the beautiful picture taken by the Swedish nature photographer Pål Nils Nilsson (Fig. 24).

Standing waves can also occur in blood which can be regarded as a suspension of blood cells in plasma. The phenomenon was first described by Dognon & Biancani (1937) who also found that blood is haemolysed by ultrasound. We found that when we submerged the tip of the ultrasonic transducer in a drop of blood both haemolysis and standing wave formation occurred. In continued experiments with our new apparatus we were able to show that *in citrated blood diluted with approximately equal*

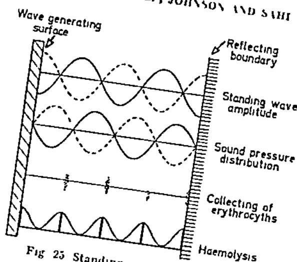


Fig 25 Standing wave relationships

quantities of physiological saline and dropped onto a glass slide, standing waves regularly occurred (Fig 21, colour picture fol p 34) Apart from standing waves, surface waves are also formed in the drop of blood when the ultrasonic wave reaches the interference surface fluid-air The whole thing looks as though the fluid was boiling Looking in the otomicroscope, it has been possible for us to see that the erythrocytes are largely collected in the nodes of the pressure waves (Fig 25 after Pohlman) At the same time there is a haemolysis, presumably also in the pressure nodes, where, as the result of acceleration, stresses on the red blood cells are greatest Standing wave formation is apparent from 0 2–1 0 watt For this reason, we use *this glass slide standing wave blood test when operating as a very simple and rapid check that the apparatus is functioning*

We have also found that ultrasonic waves can be observed in the operating cavity in Meniere operations (Zeiss operating microscope, 10 ×) A clear pulsing movement occurs in a minimal drop of fluid round the tip of the transducer, applied against the labyrinth If the fluid around the tip is mixed with a small quantity of blood clear standing wave formation occurs mainly in front of the tip of the transducer *In this way, the ultrasonic beam becomes visible and it can be aimed in the desired direction*

CHAPTER VI

EXPERIMENTS ON ANIMALS

Previous Investigations

The effect of ultrasound on the inner ear of animals has been studied by several workers, mainly histologically but to some extent also physiologically.

Vyslonzil (1949) was probably the first to irradiate the labyrinth more or less with the idea of destroying its function. Ultrasound was given extratemporally in a direction which avoided the air filled bulla, in doses which were 40 to 60 times higher than that subsequently used therapeutically. The animals which were tested with caloric and rotatory tests after irradiation showed varying degrees of vestibular lesions—from impaired function to complete abolition. Hyperaemia and nerve changes in the labyrinth could be shown histologically.

Krejci (1952) was the first to irradiate the labyrinth via the opened bulla, the air of which otherwise would have constituted an effective obstruction to the transmission of ultrasound. He used an ultrasonic transducer with a narrow glass tip (for further description, see p. 20) and irradiated guinea pigs with 4 watts/cm², using a frequency of 800 kc/s for an average of 2 minutes. By means of rotation tests and simultaneous electronystagmography he was able to show clear signs of impaired or abolished function. On the other hand the cochlear potentials were normal, indicating normal hearing. Subsequent histological studies showed severe hyperaemia and haemorrhage in the perilymphatic space, while, on the other hand the ampullae, maculae and cochlea were normal. These findings contrast with his own results of functionally completely destroyed labyrinths. Krejci tried to explain this by saying that apparently normal sensory cells do not guarantee normal functions.

Completely opposite results were described by de Stefani in 1956. Using the Italian Federici apparatus, he irradiated dogs through the opened bulla, after which the animals developed first an "irritative" and then a "paralytic" nystagmus. The dose was 10 watts/cm² for 15 minutes at a frequency of 800 kc/s. The animals were killed 8 to 14 days after irradiation. The sensory epithelium on the ampullary crista had severely degenerated with vacuolation and pyknosis of the nuclei but the cupula was intact. In the semicircular canals there were signs of coagulation of the endolymph but

there were no changes in the walls of the membranous canals. There were minor haemorrhages in the perilymphatic space with here and there detachment of the periostium. De Stefani also carried out interesting experiments on the effect of ultrasound on healing of experimental fistulae in the bony labyrinth and also in other bones. Ultrasound appeared to prevent the formation of new bone.

Arlagno in 1960 used the same technique on monkeys. One week after irradiation histological investigation showed oedema, precipitates and pyknosis. After three weeks, there was detachment of the epithelium, vacuolation and pyknosis, while finally after six weeks there was advanced degeneration of the neuro-epithelium and the cupula.

Both the investigations of de Stefani and of Arlagno seem to indicate that there is a progressive degeneration in the labyrinth. This observation is in good agreement with the clinical findings of Lumsden (1958) that short mild attacks of giddiness can occur during the first weeks after irradiation. This suggests more a progressive degenerative process than an immediate totally destructive one.

During the last few years there has been great interest in Great Britain in the histological effects of ultrasound. Brain and co-workers (1960) published a detailed study based on eight cats irradiated with a Federici apparatus. *The dosage consisted of a progressive increase up to 8 watts/cm² at* the end of 5 minutes increasing up to 10 watts/cm² at the end of 10 minutes, the total duration of application being 15 minutes. The earliest changes noted in the treated ear were vasodilatation associated with increased capillary permeability. The latter was obvious from the appearance of protein exudate in the fluid spaces of the inner ear. Exudation of protein was most marked in the vestibular part of the inner ear. After 48 hours there were, in addition, severe degenerative changes in the neuro-epithelium of the labyrinth. The changes increased with survival time and after 20 days the sensory epithelium of the crista ampullaris and the maculae had largely disappeared and the remaining epithelium showed pronounced degeneration. The ampullary nerve in one cat showed mucoid degeneration. In all the animals there were changes in the cochlea completely analogous with those in the vestibular labyrinth. These investigations further underline the progressive tendency in degeneration.

McLay, Flinn & Ormerod (1961) using a new apparatus treated three guinea pigs with an intensity of 0.9 watt/cm² at a frequency of 1350 kc/s for 30 minutes. The tip of the transducer had a diameter of ¹/₄ in, which for animal experiments and also clinical use is particularly large. A drop of saline was placed between the superior vertical canal and the transducer as a coupling medium. In one animal which was killed immediately after irradiation there were no histological changes in the inner ear. In another animal which was sacrificed after seven days there was a severe lesion in the inner ear. The membranous labyrinth had collapsed, the sensory epithelium in the cristae and the maculae was considerably degenerated

as well as in the cochlea Furthermore, there was atrophy of the stria vascularis and the underlying connective tissue Finally, lesions of the facial nerve could also be shown The third animal died four days after irradiation

In experiments on sheep using their new 3 mc/s apparatus, Angell James and co-workers (1963) have shown damage to the hair-cells both in the vestibular and cochlear end-organs There were also changes in the perilymphatic spaces where haemorrhage and increase in syncytium are apparent Their results are, however, not yet fully reported

It will be observed that previous experiments on the effect of ultrasound on the labyrinth of experimental animals are few and the results in the majority of cases are based on relatively few animals There do not seem to be characteristic changes for ultrasound *However, there is always an initial hyperaemia and oedema later followed by progressive degeneration of the neuro-epithelium in both the vestibular and cochlear parts of the inner ear*

There is only one case published, where a human labyrinth was studied after ultrasonic irradiation Beck (1958) has described a patient with Meniere's disease, in whom three weeks after ultrasonic treatment without result, a labyrinthectomy was performed by Cawthorne's method with removal of the lateral membranous canal The neuro-epithelium on the crista was severely damaged and proliferation of connective tissues was seen in the crista The walls of the membranous canal were thickened

Subsequently, Beck (1959) studied the effect of ultrasound on the cochlea of the guinea pig He used a transducer with a very fine tip, the area of which was only 1 7 mm² This was applied directly to the cochlea and the dosage was 3 watts/cm² and 6 watts/cm² each lasting for two minutes With the lower dose there were milder and reversible changes, on the other hand, with the higher dose there was pronounced degeneration

Before we began the clinical treatment of Meniere's disease, we tested our apparatus in a series of animal experiments These were then continued parallel to the clinical investigation and our aim has been to try to find out the biological mechanism behind our own and other's favourable clinical results *We have also tried as far as possible to correlate the findings in the vestibular tests to the microscopical picture to get as good an idea as possible of the effect of ultrasound*

Our Experiments on Pigeons Anatomy

Pigeons were used as experimental animals, partly because the labyrinth is easily accessible and partly because they react clearly to rotatory stimuli

The anatomy of the inner ear of the pigeon is described in detail in the classical work of Retzius (1884), from which Fig 26 is taken The three semicircular canals are clearly seen in the porous bone under a thin

there were no changes in the walls of the membranous canals. There were minor haemorrhages in the perilymphatic space, with here and there detachment of the periostium. De Stefani also carried out interesting experiments on the effect of ultrasound on healing of experimental fistulae in the bony labyrinth and also in other bones. Ultrasound appeared to prevent the formation of new bone.

Arragno, in 1960, used the same technique on monkeys. One week after irradiation, histological investigation showed oedema, precipitates and pyknosis. After three weeks, there was detachment of the epithelium, vacuolation and pyknosis, while finally, after six weeks, there was advanced degeneration of the neuro epithelium and the cupula.

Both the investigations of de Stefani and of Arragno seem to indicate that there is a progressive degeneration in the labyrinth. This observation is in good agreement with the clinical findings of Lumsden (1958) that short, mild attacks of giddiness can occur during the first weeks after irradiation. This suggests more a progressive degenerative process than an immediate totally destructive one.

During the last few years, there has been great interest in Great Britain in the histological effects of ultrasound. Brain and co-workers (1960) published a detailed study, based on eight cats irradiated with a Federici apparatus. The dosage consisted of a progressive increase up to 8 watts/cm² at the end of 5 minutes, increasing up to 10 watts/cm² at the end of 10 minutes, the total duration of application being 15 minutes. The earliest changes noted in the treated ear were vasodilatation, associated with increased capillary permeability. The latter was obvious from the appearance of protein exudate in the fluid spaces of the inner ear. Exudation of protein was most marked in the vestibular part of the inner ear. After 48 hours there were, in addition, severe degenerative changes in the neuro-epithelium of the labyrinth. The changes increased with survival time and after 20 days the sensory epithelium of the crista ampullaris and the macula had largely disappeared and the remaining epithelium showed pronounced degeneration. The ampullary nerve in one cat showed mucoid degeneration. In all the animals there were changes in the cochlea completely analogous with those in the vestibular labyrinth. These investigations further underline the progressive tendency in degeneration.

McLay, Flinn & Ormerod (1961), using a new apparatus, treated three guinea pigs with an intensity of 0.9 watt/cm² at a frequency of 1350 kc/s for 30 minutes. The tip of the transducer had a diameter of ³/₄ in, which for animal experiments and also clinical use is particularly large. A drop of saline was placed between the superior vertical canal and the transducer as a coupling medium. In one animal, which was killed immediately after irradiation there were no histological changes in the inner ear. In another animal which was sacrificed after seven days, there was a severe lesion in the inner ear. The membranous labyrinth had collapsed, the sensory epithelium on the cristae and the maculae was considerably degenerated

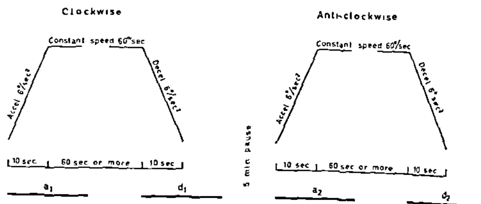


Fig 27. The principles behind the AD test (acceleration deceleration test) a_1 and a_2 nystagmus due to acceleration, d_1 and d_2 nystagmus due to deceleration There is a 5 minute interval between clockwise and anti-clockwise rotation (From Stahle, Acta Soc Med Upsal., 1957)

secretory function. In mammals there are anatomically great similarities between the stria vascularis and planum semilunatum as regards the epithelium and subepithelial vascular network (Saxen, 1948; Wittmaack, 1956) and these areas are thought to produce endolymph in the cochlea and the membranous labyrinth respectively (Saxen, 1948). Using S³⁵ Dohlman (1959) has shown in pigeons a rich secretory function from the cells on planum semilunatum According to Ormerod (1960) there is a rich secretion of sulphomucopolysaccharides from Reissner's membrane, which has abundant knot-like projections, and also from the epithelium in the saccule and utricle

The normal anatomy of the inner ear of the pigeon is shown in Figs 32, 35, 37 and 39

Labyrinthine Test and Nystagmography

The reactions of pigeons to weak rotatory stimuli before and after ultrasonic treatment were tested In contrast to mammals, birds have highly developed labyrinthine reflex pathways to the neck muscles As a result of this, they develop a clear head nystagmus, which is considerably easier to study and record than their eye nystagmus In order to [illegible] optokinetic nystagmus, visual stimuli were eliminated by means of a [illegible] black cap with a gap anteriorly for the beak, which is put on the head of the pigeon

Function was tested in a rotating chamber by means of the [illegible] (acceleratory–deceleratory test) This rotation test was described [illegible] of us (Stahle) in an earlier work in 1957 The test, which is shown [illegible] matically in Fig 27, is briefly as follows The chamber acce[illegible] 6°/sec² up to a speed of 60°/sec, where it is kept at a constant [illegible]

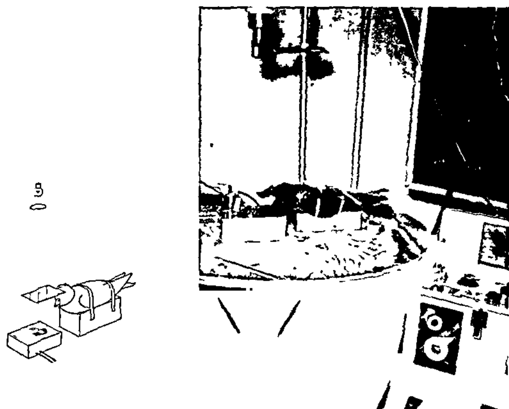


Fig 28 Rotatory test on pigeon with recording of the pigeon's head nystagmus Under the head of the blindfolded pigeon is placed a photocell which registers the rythmical variations in the lighting (From Aschan, Bergstedt, and Stahle, Acta Soc Med Upsal., 1955)

about 60 seconds, then follows a retardation with the same stimulus, 6°/sec- down to a stop After a pause of about 5 minutes a new rotation is performed, now in the opposite direction In this way four equally strong stimuli are applied and four periods of head nystagmus are obtained, beating to the right in two, and to the left in two which are then compared To a certain extent, the results are normally symmetrical Duration, number of beats as well as amplitude and eye speed of the slow phase can be calculated

During the rotation test the pigeon is held immobile in a special holder according to Ewald (1892) and placed immediately above the axis of rotation With the abdomen downwards, the animal develops horizontal nystagmus according to the Flourens-Ewald law

Nystagmus is recorded using the method described by Aschan, Bergstedt & Stahle (1955) and the arrangement for the experiments is shown in Fig 28 Above the head of the animal is placed a lamp, while below the head is a photocell A very light piece of cardboard, about 3 × 4 cm in size is *put horizontally into the cap, which is placed over the head of the pigeon* to blindfold it This piece of cardboard casts a shadow over the photocell

ument (Mingograph,

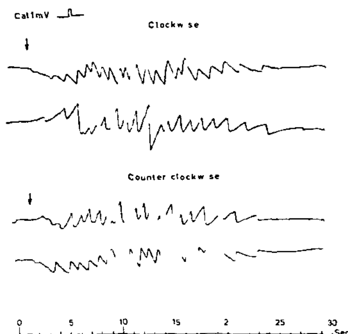


Fig 29 Nystagmographical recordings of the pigeon's head nystagmus in a rotatory test which gave normal results pigeon No 15 The principles behind the AD test appear in Fig 24
The directions for the four nystagmus reactions are beginning at the top right beating left beating left beating and right beating The arrows indicate the start of acceleration and deceleration

Model 12 Elema Schonander AB Solna Sweden) In this way, the head nystagmus of the animal is recorded directly on paper The reliability of the method has been tested previously (Aschan and co workers 1955) a comparison was made between nystagmographical recordings from rabbits (Schott's method) and pigeons (photocell technique) After having been given the same stimuli they showed agreement

An AD test from an untreated animal is shown in Fig 29 The four tracings show that every nystagmus response lasts about 25 seconds so that there is good symmetry The uppermost tracing in the figure is recorded during acceleration to the right and the nystagmus beats to the right in the next recording taken during the subsequent deceleration the nystagmus beats to the left The third tracing again shows left beating

nystagmus as the result of counter-clockwise acceleration. Finally, the lowest tracing shows right-beating nystagmus on deceleration

The duration of nystagmus and the number of beats normally show great individual variation The animal shown in Fig 29 had vigorous reactions Other pigeons had nystagmus for only 10 to 15 seconds

Attempts were made to test hearing with a Galton whistle at frequencies of about 2000–8000 c/s The animal can develop a withdrawal movement in the neck muscles from this, but the response is inconsistent. At the present stage, we do not feel that we can draw any conclusions concerning cochlear function on the basis of these very primitive tests. Measurement of the cochlear microphonics are planned

The Effect of Irradiation

Thirty pigeons were irradiated with ultrasound Before irradiation the labyrinth was dissected out under local anaesthesia, the head of the pigeon being fixed in a ring shaped holder as described by Ewald (1892) During the operation, an otomicroscope has been used with a magnification of 6 × to 16 × Irradiation was directed mainly against the ampullary parts of the semicircular canals However, the technique has varied somewhat The time of irradiation was from 30 seconds to 5 minutes and the power 1 0 watt The diameter of the tip of the transducer has been 2 2 mm [1] A drop of saline was applied to the tip of the transducer in order to give good contact with the labyrinth The transmission of ultrasound can be seen the whole time in the microscope in the form of small wave movements in the fluid coupling Ultrasound gives no immediately visible changes in bone, blood vessels or muscle in the operating area

In a number of animals, a vigorous eye nystagmus was observed during irradiation, first towards and then away from the irradiated side We have not been able to study head nystagmus during the actual irradiation because at this time the head was fixed Immediately afterwards the animals showed abnormal vestibular reactions with impaired balance, turning of the head towards the operated side and, in isolated cases, spontaneous head nystagmus beating from the operated side ("paralytic" nystagmus)

The functional and histological changes vary with the dose of ultrasound given With lower doses, some function remains, but with higher doses this can be completely eliminated Two typical examples of this follow

Pigeon No 15 Preoperatively, at the rotation test, normal nystagmographic recordings were obtained as shown uppermost in Fig 30 The animal was irradiated on both sides with an interval of two weeks, first

[1] In later and present experiments a transducer was used with a tip diameter of 1 5 mm which could be applied directly to the wall of the ampulla The power and treatment time have been raised in several experiments

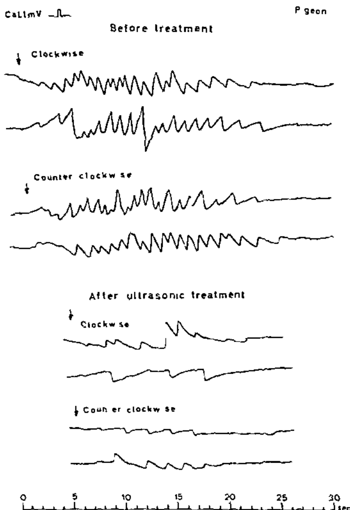


Fig 30 Rotatory tests on pigeon No 15 Before bilateral ultrasonic irradiation—normal reactions The animal was irradiated first on the left and then on the right side with an interval of two weeks The rotatory test three weeks after the last irradiation shows greatly impaired function (the four lower tracings)

on the left and then on the right The dose was 1 watt and irradiation time 45 seconds towards each of the three semicircular canals The day before the last operation a rotation test showed a suggested directional preponderance to the right, which could be understood as a sign of impaired function of the left labyrinth Three days after irradiation of the right side, a right-beating spontaneous head nystagmus was observed A rotation test carried out three weeks after the last irradiation showed greatly impaired function, possibly with some directional preponderance to the right (the undermost tracings in Fig 30)

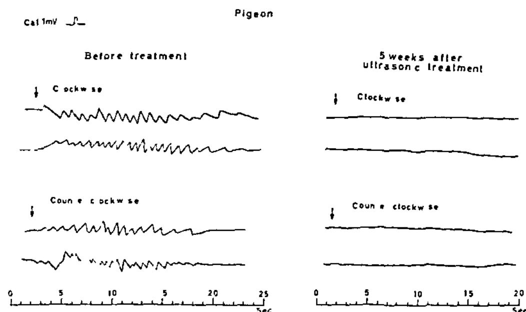


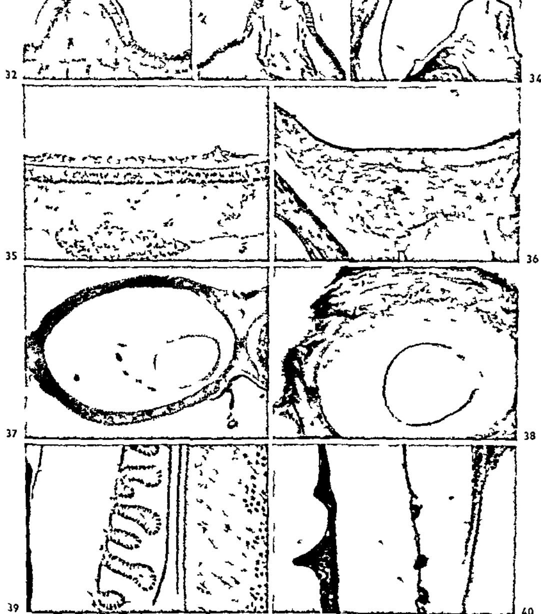
Fig. 31. Rotatory tests on pigeon No. 4. Before bilateral ultrasonic irradiation—reactions normal. Five weeks after treatment no reactions can be elicited. The arrows indicate the start of acceleration and deceleration. The principles behind the AD test are shown in Fig. 2.

The animal was sacrificed three weeks after the last irradiation. Histological examination revealed relatively small changes in the labyrinth. The sensory epithelium in the cristae was partly degenerated with vacuolization and pyknosis of the nuclei. The normally clear cell differentiation on the ampullary cristae had partly disappeared. The cupulae were collapsed and shrunken. The walls of the bony semicircular canals were here and there thickened with a suggestion of proliferation of connective tissue in the perilymphatic space.

In this case ultrasonic irradiation produced partial disappearance of function of the labyrinth and the anatomical and functional findings complement each other well.

Pigeon No. 4. Irradiation was carried out bilaterally with an interval of two weeks and a power of 1 watt for 4 minutes, the beam being directed mainly towards the ampulla of the lateral semicircular canal and the vestibule. A rotation test before operation showed symmetrical reactions with a duration of about 20 seconds after every stimulus (Fig. 31). Renewed nystagmography five weeks after the last irradiation showed that nystagmus could no longer be elicited, as shown in the right part of Fig. 31. The animal was sacrificed five weeks after irradiation.

The histological examination of pigeon no. 4 showed very severe damages



Figs 32-40 The inner ear of the pigeon—non treated and irradiated animals

Fig 32 Normal crista cupula and normal secretory epithelium around the base of the crista from a non treated animal

Fig 33 A section from a pigeon hav g received a small dose of ultrasound showing minor damage t the cupula neuro epithel um and secretory epithelium (centre)

Fig 34 Severe lesions due to strong ultras nic irradiation are seen in both the neuro ep thel u a d secretory epithel u arou d the base of the crista and on the planum em lu atum Serious ascular damage is also present (pigeon No 4)

Figs 35 36 A n r al utr cl to the left and a severely damaged one to the right (pigeon No 4

Figs 3 -38 A normal semicircular canal to the left and the partly obliterated one to the right

Figs 39-40 T the left a rmal lagena from a non treated animal and to the right from a an al rradiated with large doses of ultrasound The figures show from the left the foo plate of the c lumella Reissners membrane with its secretory epithelium the neu f um and the nerve cells The neuro-ep thelium as well as the secretory ep thel u

both in the vestibular and in the cochlear parts of the inner ear (Figs. 34, 36, 38 and 40 in colour) The neuro-epithelium in the cristae and the maculae was completely destroyed and only fragments of the basal cell layer remain The cupula and the otolith of the utricle had disappeared completely The secretory epithelium on the planum semilunatum in the lateral ampulla had largely disappeared In the cristae and in the subepithelial zone of the utricle there was in some areas proliferation of connective tissue In other large areas, on the other hand, the connective tissue had completely disappeared and instead homogeneous, structureless masses could be seen, which consisted of mucous oedema, known as *myxomatous degeneration*, which on specific staining with toluidine blue showed metachromasia The capillaries were greatly dilated and thromboses were present The nerve fibres had broken up, while here and there nerve tissue had been replaced by vacuoled xanthoma cells A cross-section of a semicircular canal (Fig 38) showed advanced changes in the walls with callus like formation of new bone This seemed to have been mostly an endosteal reaction The perilymphatic space was completely obliterated by newly formed tissue of connective-tissue type The walls of the membranous canals were somewhat thickened but the canals were still open

Equally severe changes were seen in the lagena (Fig 40) The neuro epithelium on the basal membrane and the ganglion cells were severely degenerated The acoustic nerve was damaged The normally abundant secretory epithelium on Reissner s membrane had largely disappeared, and remaining cells showed degeneration

It will be seen that in this case ultrasound functionally eliminated the labyrinth which was supported by the extremely severe anatomical lesions

A summary of the histological and functional changes observed in our experimental studies of animals reveals the following effects of ultrasound

Histological

1 Degeneration of both the neuro epithelium and the secretory epithelium in the ampullae, utricle and saccule

2 Collapse or decomposition of the cupula

3 Degeneration to varying degrees of both the neuro-epithelium and secretory epithelium in the lagena (the pigeon's cochlea)

4 Changes in the wall of the bony labyrinth—in the most pronounced cases, callus-like formations

5 Varying degrees of obliteration of the perilymphatic space as a result of formation of new tissue

6 Thickening of the walls of the membranous labyrinth

Functional

7 During irradiation homolateral or "irritative", in some cases followed by contralateral or "paralytic" nystagmus

8 After irradiation impaired vestibular function

CHAPTER VII

CLINICAL APPLICATION OF OUR APPARATUS AND RESULTS

Patient Material. Different Clinical Test Methods

In the Oto-Laryngological Department in Uppsala we have so far treated 50 cases of Ménière's disease, of which 26 were women and 24 men. The average age was 48 years. The length of illness before operation was on the average eight years—varying from one to 21 years. Before operation every patient had undergone medical treatment of different types.

All of the cases irradiated had been greatly disabled by frequent attacks of giddiness. The majority had had a severe impairment in hearing of the neural type. Signs of recruitment phenomenon were found in all cases when Békésy audiometry, Fowler's balance test and impedance measurements were carried out.

Careful assessment of vestibular function was made before and after operation by means of *electronystagmography*, which technique has not previously been applied to this special field. This method allows an objective analysis of spontaneous and positional nystagmus as well as calorically and rotatory induced nystagmus. A numerical evaluation of the labyrinth function can be quickly made from the nystagmographic registrations and functional changes caused by the ultrasound can be measured with rather good accuracy. We believe we have in this way increased the possibility of judging the ultrasonic effect and at the same time we are in a good position with regard to the important correlations of labyrinthine symptoms and signs. Positional and caloric tests with simultaneous nystagmography were performed in all cases, but rotatory tests were performed only on a small number.

Preoperatively spontaneous or positional nystagmus was found in 13 cases. The caloric test showed diminished excitability in 21 cases, directional preponderance in 7 cases and normal function in 19 cases. Two bilateral cases have been treated on the side which showed the most severe hearing loss.

The caloric test has been performed with the patient lying on a couch with the head raised at an angle of 30°. Water at 30° C and 44° C according to Fitzgerald & Hallpike (1942) has been used and the syringing time has been 30 seconds. The calorically induced nystagmus has been estimated on the basis of the duration and the maximum intensity. Stress has been

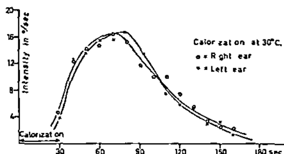


Fig 41 The intensity of the reaction of a normal subject to syringing at 30°C Each dot represents the mean for a 10 second period The *maximum intensity* is represented by the dot at the top of each curve (From Stahle, Acta Soc Med Upsal., 1956)

laid chiefly upon the maximum intensity, which is a mean of the speed of the slow nystagmus phase during a ten second period at the peak of the reaction (Stahle, 1958) The maximum intensity is illustrated in Fig. 41 The difference between right and left ear and right- and left-beating nystagmus has been estimated according to Jongkees *et al* (1962) with figures based on the normal material published by Stahle (1958) The normal limits for the duration and the maximum intensity when comparing right and left ear is set at 10% and 20% respectively These figures exceed somewhat twice the standard deviation

In addition, all patients underwent medical, neurological and ophthalmological examination before operation The cell system of the ear and the internal auditory meatus were X-rayed In most cases, EEG was also performed Only those cases in which the diagnosis of Meniere's disease was clear according to current criteria were selected for irradiation

Technique of Operation

On the whole we have followed Arslan's technique (1953) and his last modifications (1962) with local anaesthesia, retroauricular incision and transmastoidal approach (Fig 42) The bony labyrinth is dissected free under the microscope It is particularly important that both the horizontal and vertical semicircular canals be exposed if possible, in order to obtain a good surface on which to place the transducer The incus is left intact As far as possible, exposure of the dura has been avoided in order to prevent possible transmission of the ultrasound into the cranial cavity

The bony wall of the labyrinth is thinned out with a diamond drill on the posterior part of the horizontal canal until the "blue or grey line" is just visible In the last 20 cases of our series, furthermore, we have thinned out the bony wall down in the spongeous enchondral bone above and behind the ampule of the lateral canal in the angle between this and the

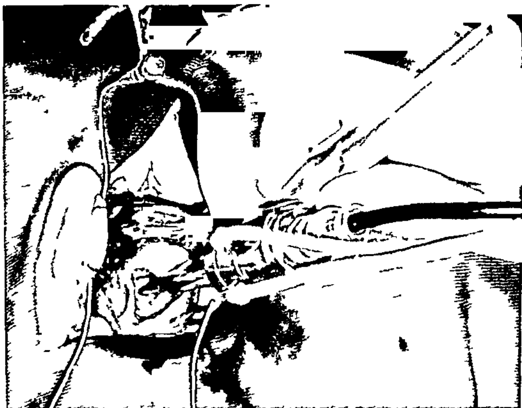


Fig. 42. The transducer applied to the labyrinthine wall. Thanks to the fine tip one has a good view of the field of operation.

superior vertical canal (Fig. 43), thus making a groove affording a good contact area for the tip of the transducer. The wall then remaining should be about 0.5 mm thick. Optimal conditions now exist for the ultrasound to penetrate the bone with a minimum absorption loss (cf. above p. 35).

Pethidine and atropine were given as premedication. Antibiotics (penicillin and tetracycline) were started two days before operation and continued for a total about 8 days. From the time of operation the patient remained in the hospital for about two weeks.

In this connection we would like to stress the importance of avoiding such drugs causing a damping of the function of the vestibular system, for example antihistamines. Such drugs could bring about a condition in which no nystagmus could be elicited by the ultrasound, thus making the assessment of the optimal dosage of irradiation impossible.

The Technique of Irradiation. The Labyrinthine Reactions to Ultrasound

Before the labyrinth is irradiated the effect of the ultrasound is tested on a drop of blood mixed with saline (the glass slide test — cf. Fig. 21, colour picture). The observation of surface waves must be considered



Fig 43 The ultrasonic approach to the labyrinth in man The tip of the transducer is placed into the grove behind and above the lateral semicircular canal

a very rough test of the equipment s functioning Relative estimates of intensity can be made from these simple tests through experience and serve as a simple guide or check that the treatment head is delivering ultrasound at the proper level This assures the operator that no malfunction or misadjustment has changed the intensity level to a large degree over that expected

Before application to the labyrinth the tip of the transducer is dipped in physiological saline solution so that a small drop is attached to the tip When the ultrasound is switched on the drop can be seen in the microscope (10 ×) to rise that is to be actually set in vibration and be strikingly adherent to the tip of the transducer This drop of fluid, sometimes mixed with a small amount of blood from the operation cavity, is used as a coupling medium between the transducer and the wall of the labyrinth so that good transmission of ultrasound is obtained The tip, coupled in this way is directed against the blue line of the lateral canal and especially against the groove above and behind the lateral canal thus avoiding the facial nerve With this technique it can probably be said that the ultrasonic beam will reach the otoliths as well as the ampullae The last mentioned application is illustrated in Fig 43 which figure also gives an excellent picture of the size of the tip (2 6 mm in diameter)

In the coupled drop and the adjacent small collection of fluid, the

ultrasound can be observed in the microscope as pulsating small light reflexes and often also as fine standing waves of blood. At an output above 2 watts a clear "fog" can occur in the form of smoke-like, small drops around the tip. *By means of this visualization of the ultrasound in the operating cavity, we have succeeded in developing a new irradiation technique. This allows us to direct the ultrasonic beam so that irradiation of the facial nerve and the cochlea can be avoided. It is now possible for us to see exactly where the ultrasound penetrates into the labyrinth.*

Irradiation is usually begun with a power of about 1 watt (=20 watts/cm²). It is then gradually increased until we have reached the power with which a nystagmus of second to third degree can be observed and recorded, directed towards the irradiated ear (homolateral or "irritative" nystagmus). This dose is usually 1.0–1.5 watts (20–30 watts/cm²). If it is possible, without causing too severe nausea, we raise the power to 2 watts (40 watts/cm²). Irradiation is continued as long as homolateral nystagmus can be elicited. This period is called *the irritative phase*. A short pause in the irradiation is taken approximately every third to fourth minute in order to check the apparatus and to "couple" a new drop to the tip of the transducer. Good fluid contact with the labyrinthine wall is necessary to obtain any effect. Even the most insignificant layer of air between the flat tip of the transducer and the labyrinth can interfere with transmission of the ultrasound. On the other hand, under no circumstances may fluid or blood completely cover the cavity of the horizontal canal and come into the middle ear or fill up the jugo-digastric area. Ultrasound can with absolute certainty be transmitted in such a fluid layer and may thus cause damage to the facial nerve.

After irradiation for 10, 15, or 20 minutes, it can usually be observed that the "irritative" nystagmus gradually disappears and is replaced by irregular, sometimes wavelike eye movements. This is what is known as *the intermediate phase*, which can last about five minutes or can be very short. At this point, we usually increase the power to 2–2.5 watts to find out if the function can be eliminated. If the "irritative" nystagmus continues, irradiation is carried on intermittantly with the higher dosage. The irritative phase can last up to 40 minutes, as shown in Fig. 16. Following the intermediate phase, a weak nystagmus is sometimes seen directed from the irradiated ear, contralateral or "paralytic" nystagmus, which has been characterized as *the paralytic phase*. Arslan (1953, 1958, 1962) *gives this as the sign that the destruction has been accomplished*. As soon as we have observed and recorded definite signs of the paralytic phase, irradiation is stopped.

In a number of cases repeated caloric tests with cold and hot water poured into the operating cavity, were carried out during the operation to determine whether or not the labyrinth was paralyzed. However, it may be difficult to assess the effect of calorization because of the reversal of nystagmus due to the ultrasound (cf. Electro-nystagmography during ir-

radiation, page 58). Furthermore, the effect of ultrasound is said to be progressive, and we do not know if it is desirable, or even possible, in every case to completely extinguish the caloric excitability on the operating table. For these reasons, we consider the value of the caloric test during irradiation up to now to be limited.

Following irradiation, the wound is closed primarily, and a small rubber drain is left in the lower end of the incision. The patients have remained in bed for one to two days, after which time they have been increasingly mobilized. *Postoperative symptoms of nausea and giddiness have been insignificant throughout.*

The average irradiation time has been 32 minutes. The longest time being 50 minutes and the shortest, 10 minutes. The average figure for the total power used is 2600 joules, the highest dose being 5500 joules and the lowest 800 joules.

We have found it desirable up to now to have a physicist or a trained technical assistant in the operating theatre during the whole irradiation.

Electronystagmography during Irradiation

Electric recording of the nystagmus during the ultrasonic irradiation was done for the first time at the Uppsala clinic. This means that we can follow the nystagmus at every moment throughout the whole procedure, and get information not only of the direction, but also of the intensity (eye-speed of the slow phase, frequency, amplitude, etc.). The operator gets an immediate and exact analysis of the effect of irradiation at every moment.

Electro nystagmography is carried out according to a method described in detail by Aschan and co-workers (1956) and which involves the recording of displacements of the corneo-retinal potentials. Electrodes are placed for recording of horizontal nystagmus but in some cases also for vertical. Since the eyes are observed at the same time through Frenzel's glasses by an assistant, it is not possible to have the eye-lids closed (Fig. 44).

During the first operations we had difficulty in obtaining good recordings because of interference from other electrical equipment. However these problems were eliminated after systematic grounding and coupling by means of a system similar to that used by Aschan in animal experiments. In principle, this is a preamplifier with a cathode follower input effectively producing a good interference shield.[1] This cathode follower is seen in Fig. 44 as a small rectangular box from which electrodes go to the face of the patient.

A series of nystagmographic recordings taken during operation are shown in Figs. 45, 46, 47. An upward deflection in the tracing indicates eye move-

[1] The apparatus was built by Mr. H. Johansson, Aeromedical Laboratory, Department of Oto-laryngology, Uppsala.

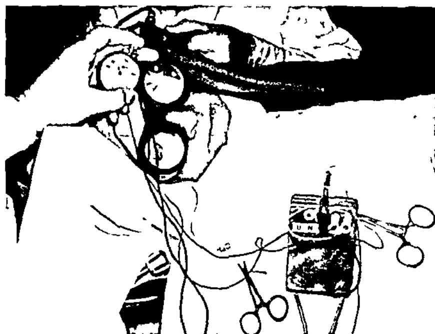


Fig. 44. Electronystagmographical recordings are always done during the entire irradiation. *The cables from the skin electrodes are seen in the picture and go to the interference shield which is enclosed in the square box to the right of the picture.*
Furthermore the patient's eyes and face are observed by an assistant. Frenzel's glasses are used.

ments to the right, while a downward deflection in the tracing indicates eye movements to the left. The calibration is for about 10° eye movement. Unless stated to the contrary, the paper speed is 10 mm/sec.

The electronystagmography has facilitated and made possible a number of interesting observations. When the ultrasound is directed towards the horizontal semicircular canal and its ampulla, the nystagmus is mainly horizontal. Vertical, rotatory and diagonal nystagmus may occur if the beam is directed towards the vertical canals. In other words, the nystagmus induced by ultrasound, beats in the plane of the irradiated semicircular canal. This observation, never before made in man, is further evidence of the validity of the Flourens-Ewald law which states that "nystagmus beats in the plane of the stimulated canal". This result confirms earlier observations made by one of us (Stahle, 1954) on the pigeon.

When the tip of the transducer is placed in the groove above and behind the horizontal canal and the beam is directed towards the vestibule, the "irritative" nystagmus is predominantly horizontal. The "paralytic" nystagmus has always been mainly horizontal.

The nystagmus during a full course of irradiation may differ from case to case. One main difference is the length of the irritative and intermediary phases, another is the intensity of the nystagmus. Two examples of this follow.

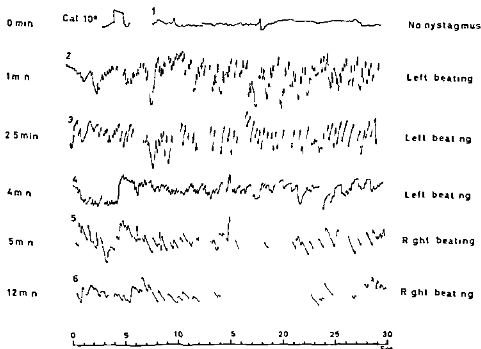


Fig 45 Case No 35 Example of a quickly produced destruction After 1 minute s irradiation of the left labyrinth can be seen a brisk, left beating nystagmus (irritative) After 5 minutes irradiation the nystagmus had changed direction to right beating (paralytic) Further left beating nystagmus could not be elicited in this case Irradiation time 13 minutes Total output 800 joules Paper speed 10 mm sec

Fig 45, which illustrates case No 35—a 30 year old man with a left sided Meniere's disease—shows a very brisk homolateral nystagmus during the first minutes of irradiation This irritative phase has a duration of about $4^1/_2$ minutes followed almost immediately by the paralytic phase, where a typical contralateral nystagmus is recorded The average speed of the slow nystagmus phase in the irritative phase (tracing No 2) is 40°/sec and in the paralytic phase (tracing No 5) 9°/sec The irradiation time was 13 minutes and the total dose given 800 joules, which values lie far below the average In spite of the small dosage this man has been almost free from vertigo and the caloric test showed a greatly reduced excitability at follow up

The second case (No 13) illustrated in Fig 46, is that of a 60 year-old woman with a right sided Meniere s disease During the first part of the

NYSTAGMUS DURING ULTRASONIC IRRADIATION

MENIERE'S DISEASE RIGHT EAR

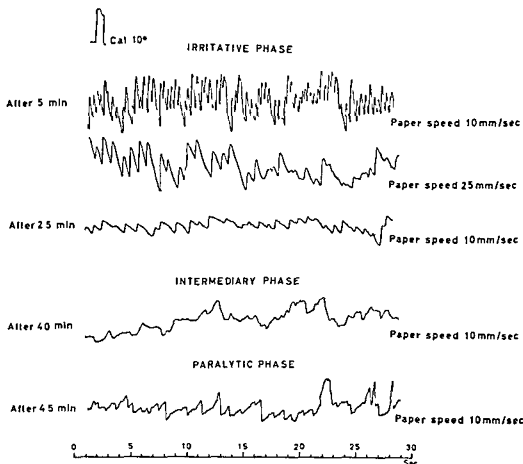


Fig 46 Case No 13 Example of nystagmus during the course of irradiation During the first part of the irritative phase, a brisk right-beating nystagmus During the intermediate phase, which came after approximately 40 minutes of irradiation, no nystagmus can be recorded The paralytic phase came late in this case, first appearing after 45 minutes characterized by a weak left beating nystagmus Irradiation time 45 minutes Total output 2700 joules

irritative phase a very brisk right-beating nystagmus is recorded with an eye speed of approximately 20°/sec (the two uppermost tracings) This "irritative" nystagmus gradually slows down The intermediary phase, during which no nystagmus can be recorded comes after approximately 40 minutes of irradiation, followed 5 minutes later by a contralateral nystagmus, indicating the paralytic phase

On repeated occasions, it has been noticed that during the irritative phase, the nystagmus suddenly completely changes direction during a

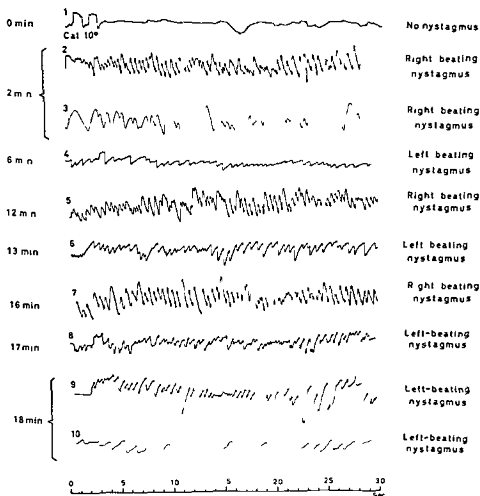


Fig. 47. Case No. 31. Nystagmus may change direction several times during the irradiation. The irradiation is interrupted for three minutes between tracings Nos. 3 and 4, 5 and 6. A clear left beating nystagmus is registered during these intervals, i.e., pseudo-paralytic nystagmus.

After between 16 and 17 minutes of irradiation a reversal of nystagmus from right beating to left beating is seen during the irradiation (tracings Nos. 7 and 8). This left beating nystagmus lasts for several hours and is taken as an indication of destruction, i.e., "paralytic" nystagmus.

Paper speed is 10 mm/sec except in tracings Nos. 3 and 10, where the speed is 25 mm/sec. Tracings Nos. 2, 3 and 9, 10 show the same nystagmus but are registered at a different paper speed. Irradiation time 22 minutes. Total output 2200 joules.

short pause in the irradiation and beats away from the irradiated labyrinth. This diametrically opposite nystagmus usually dies away after two or three minutes after which the eyes are still again. When irradiation is resumed, a typical "irritative" nystagmus returns. This phenomenon may take place several times during the course of the irradiation. The stronger the "irritative" nystagmus is, the stronger appears the following nystagmus in the opposite direction. *These momentary and repeated changes in direction of nystagmus induced by ultrasound have not been previously described and are not a sign of destruction.* They are probably an expression of a temporarily loss of function of the irradiated labyrinth following overstimulation, in which the tonic activity of the non-treated side temporarily takes over.

This reversal phenomenon is illustrated by case 39 (Fig. 47)—a 50-year-old woman with a right-sided Menière's disease. Tracings Nos. 2 and 3 show a brisk, right-beating "irritative" nystagmus during the first minutes of irradiation, after which the irradiation is stopped for approximately 3 minutes. During this pause a weak left-beating nystagmus can be recorded (tracing No. 4). When the irradiation is continued, a right-beating "irritative" nystagmus can again be recorded (tracing No. 5) with the left-beating nystagmus appearing again during the next pause. This pattern is repeated (tracing No. 7). After approximately 17 minutes, no more "irritative" nystagmus could be elicited by irradiation and the recordings showed a long-standing left-beating "paralytic" nystagmus. As can be seen directly from the recordings, the "irritative" nystagmus is much more intense than the following nystagmus in the opposite direction (cf. tracings Nos. 2–4, and 5–6). The average eye speed of the slow phase in tracing No. 2 is 27°/sec and in No. 4, 5°/sec. This nystagmus we have designated "*pseudo-paralytic*" in order to differentiate it from the true "*paralytic*", which indicates a real destruction. *This transitory direction change of the nystagmus can possibly have been interpreted by other ultrasound researchers as a sign of an accomplished destruction and thereby have been the cause of a too early cessation of irradiation. If such has been the case, it could be one explanation for the poorer clinical results of some published series.*

Similar reversals of nystagmus have been observed following violent paroxysmal positional nystagmus (Stahle, 1960), a form of nystagmus which Barany (1921) and Dix & Hallpike (1952) considered to be released from the utricle. Our observations thereby to some extent support their view of the peripheral release mechanism.

The Clinical Follow-up

A careful follow-up has been done on the first 50 cases treated using different hearing tests and electro-nystagmography. The observation time ranges between 2–20 months. In view of the relatively short observation

TABLE 2 *Results of ultrasonic treatment of Meniere's disease*

	Vertigo		Hearing			Tinnitus		
No of cases	Improved	Not improved	Improved	Unchanged	Deteriorated	Improved	Not improved	Facial paralysis
50	45	5	6	28	16	11	39	1

time in some cases, we have, in Table 2 which shows results, refrained from further subdividing the group headed 'improved' With respect to vertigo, a number of cases have been completely free from giddiness The estimation of the hearing is based on a median value for the frequencies 500, 1000 and 2000 cps in the pure tone audiometry A difference in the pre- and post-operative audiogram not exceeding ±10 db has been designated as "unchanged' An additional control with regular six-month check-ups has been started, the results of these will be presented later

A second irradiation was done in two cases which showed no relief from vertigo after the first irradiation Both of these cases have been re-examined and are now free of vertigo

In an attempt to more closely ascertain the effect of ultrasound on the neuro-epithelium of the labyrinth, a careful analysis has been made of the caloric test in the first 37 cases The results of the caloric test which will be described below, are completely based on electro nystagmography, carried out with the patients' eyes closed in a partial darkness This method is much more sensitive than the conventional non-nystagmographical methods, with or without Frenzel's glasses The reason for this being that fixation eliminates nystagmus to a large extent during investigation in a

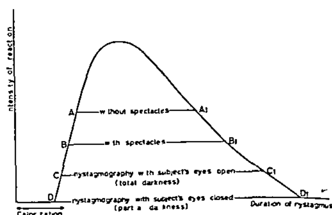


Fig 48 Diagram illustrating the inhibitory effect of fixation upon caloric nystagmus The Uppsala technique—nystagmography in partial darkness with the subject's eyes closed—give higher intensity and longer duration of nystagmus compared with other methods (From Stahle Acta Soc Med Upsal 1956)

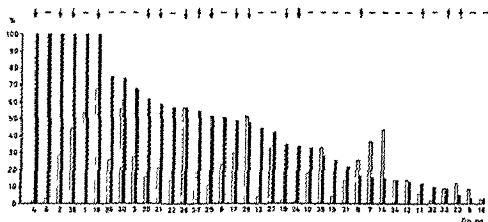


Fig. 49 Diagram illustrating the caloric reactions before and 2 months after ultrasonic irradiation as well as the post-operative hearing. The caloric reactions are assessed from the maximum intensity and not the duration Ordinate: percentage difference in right-left sensitivity, abscissa: patient number.

The white columns show the percentage difference in right-left sensitivity before irradiation and the black columns, after irradiation The difference between the white and the black columns in every single case shows the reduction in excitability due to ultrasound

The 100% post-irradiative difference in right-left sensitivity in the six cases to the left implies that the caloric sensitivity has been completely eliminated

↓ means deterioration of hearing, ↑ improvement of hearing, and — no effect upon hearing

lighted room with opened eyes—the most common procedure in all the world (Rossberg, 1954, Aschan, 1955, Stahle, 1956, *et al.*) With the Uppsala technique, on the other hand, fixation is completely eliminated and this allows us to register even a nystagmus so weak as to have been undetectable either with or without Frenzel's glasses. The inhibitory effect of fixation is clear from Fig 48, which shows that the duration and the intensity of the calorically induced nystagmus is most extensive when assessed nystagmographically with our technique *This means that cases with a reduced labyrinthine function tested conventionally would have been judged as having a completely eliminated function, but with nystagmography might have shown definite reactions*

We have compared the pre-operative caloric reactions with those recorded at follow-up two months or more after the irradiation. The comparison has been based on the maximum intensity, which is a more expressive factor than duration in the judging of calorically induced nystagmus The method used in assessing the maximum intensity and the normal values are given on page 51

The differences in right-left sensitivity, both pre-operatively and at follow-up are given in Fig 49 The figure under the columns designate the patient in question's number in the series No 1 is the first irradiated case The white column to the left gives the pre-operative difference in

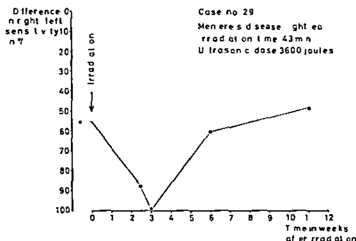


Fig 50 Example of transitory elimination of caloric excitability Before irradiation the excitability of the right ear was reduced to 55% three weeks after irradiation the caloric reaction was completely lost but after six weeks the labyrinthine function had returned and the caloric excitability reached the pre operative level Each dot represents one caloric test

right left sensitivity for maximum intensity the normal figures not exceeding approximately 20% *A definite diminished excitability is seen in 14* cases (Nos 2 38 1 18 36 30 3 21 26 9 17 29 27 and 35) The black column shown just to the right of each white column indicates the percentage difference in right left sensitivity two months or more after ultrasonic treatment In 26 cases grouped together in decreasing order to the right in the figure a canal paresis could be shown in the follow up (Nos 4–31)

Where the black column is longer than the white an additional reduction of caloric sensitivity is roughly indicated Such a reduction which would seem to imply a diminished labyrinth function has been noted in 23 cases Of the remaining 14 cases there was in 8 a normal caloric reaction before as well as after irradiation in three cases a reduced excitability before but normal after irradiation and finally in three cases a reduced excitability both before and after (Nos 26, 29 and 35)

Six cases (Nos 4 6 2 38 1 and 18) placed at the extreme left of the figure showed a difference of 100% in right left sensitivity in the follow up This is to say that the ultrasonic treatment has brought about an elimination of caloric excitability in the affected ear These cases of which 4 were among the first treated had in common that the boring out over the lateral semi circular canal was done so deeply that a minimal fissure has appeared in the endosteal bone Because of this much larger amounts of ultrasound were directed into the labyrinth in comparison with the other cases where the bony wall was intact In cases Nos 4 and 6 normal caloric reactions had been noted before the operation while cases Nos 2 38, 1 and 18 showed a reduced caloric excitability with differences from 30 70%

Case No. 13

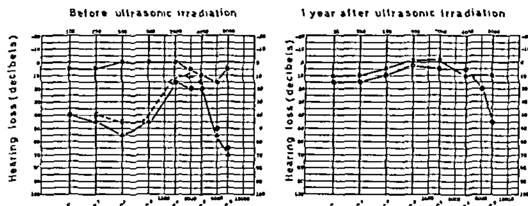


Fig. 51. Pure tone audiogram before and after ultrasonic irradiation of the right labyrinth

In 31 of the first 37 cases treated a reduced and in a few cases a normal caloric reaction has thus been elicited with 30° or 44°C water, or in isolated cases with ice water, on the irradiated side. *The conclusion from this is, that ultrasound in our dosage and with our technique does not completely eliminate the caloric excitability except in those cases where the endosteal bone is penetrated.* If the good clinical results up to now are correlated to that fact, this would seem to point out that the reduction of the function of the neuro-epithelium is not the only vital turning point, but that other factors also play a role, possibly a reduction in the secretion of the endolymph. Preliminary results of a later series, nevertheless, point out that higher dosages of ultrasound can further reduce the caloric reaction without completely eliminating it, using the strict criteria we have set ourselves.

Transitory elimination of the caloric response has been noted in some cases, an example of which is illustrated in Fig. 50. Before irradiation this patient showed a strong reduction of the caloric reaction on the affected side. The difference in right–left sensitivity (maximum intensity) was 55%. Ultrasonic irradiation was done on October 2, 1962, after which a progressive reduction of the caloric response was noted, ending with a total elimination (October 23rd). Six weeks after irradiation the caloric response had returned, and after 10 weeks, finally, the pre-operative level was again reached (December 17th). These findings correspond well to the case history which describes an improvement of vertigo. The hearing was not influenced by irradiation.

The ultrasonic effect upon hearing is simply illustrated in Fig. 49, – means no influence upon hearing, ↑ a hearing gain, and ↓ a deterioration. The limits for judging have, as previously mentioned, been set at ± 10 db expressed as a mean value for the frequencies 500, 1000 and

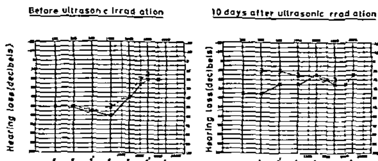


Fig 52 Pure tone audiograms before and after ultrasonic irradiation of the left labyrinth Hearing on the right side normal

2000 cps As the illustration shows, the hearing in 20 of 37 cases has not been affected, it has been worsened in 12 cases, and improved (> 10 db) in five cases In two cases we have been able to observe how a serious longstanding hearing loss has been restored to almost normal some weeks after irradiation This fact seems to rule out the possibility of a spontaneous remission, and could instead well be an expression of the effectivity of ultrasound A short description of these cases follows

Case No 13 A 60-year-old woman with a typical 11-year history of right-sided Meniere's disease, who has had a constant hearing decrease in the right ear for the past 3 years. Pre operative investigation showed a typical pure-tone audiogram of flat loss type, recruitment in the Fowler test and the impedance test The caloric reactions were normal Improvement in hearing was noted as early as 4 weeks after irradiation on the affected side and became successively better and was completely normal after 12 weeks One year after irradiation she still has normal hearing (Fig 51) Since the operation, the patient has been almost completely free from vertigo The nystagmus pattern in this case during irradiation is illustrated in Fig 46 on page 58 Irradiation time—45 minutes, ultrasonic dose—2700 joules

Case No 43 A 30-year-old man with a 5 year history of typically left-sided Meniere's disease Pure-tone audiograms had, during the last 3 years, shown a constant nerve deafness as low as 50 db in the left ear The caloric reaction was slightly diminished on the left side Ten days after the operation the hearing had become almost normal as the audiograms show (Fig 52) The caloric reaction was further reduced and the vertigo improved Irradiation time—30 minutes, ultrasonic dose—3300 joules

Facial paralysis has been noted in only one case (No 26) among the 50 treated The paralysis appeared first on the 6th day after the operation It progressed for a period of 2–3 days and was thereafter complete Three weeks after the operation, function began to return and was completely

restored after 6 weeks No operation for decompression was attempted Irradiation time in this case was 17 minutes, and the total dose of ultrasound was only 1400 joules

With the exception of the single instance of facial paralysis mentioned above, no complications have been observed Indications that ultrasound could damage the brain have never appeared

GENERAL DISCUSSION

The aim of the ultrasonic treatment of Meniere's disease should be to relieve or considerably improve the patients' vertigo and to preserve the hearing at its pre-operative level This has been made difficult by the fact that it has not been possible to estimate the optimum dosage of ultrasound and because there are so many different factors which influence the result. It seems, however, that research is succeeding more and more in defining those separate elements which are of essential importance *It has been shown that the effectiveness of the treatment depends principally upon the following factors (1) bone thickness within the irradiated area, (2) the transmission of ultrasound to the bony wall (3) the direction and focussing of the beam, and (4) the total ultrasonic energy given*

We always thin out the bony wall over the lateral semi-circular canal and also behind and above the same canal This last mentioned approach facilitates a good application of the transducer tip, which fits well into the groove made here in the enchondral bone A minute drop of saline in the groove assures good transmission and aids in directing the sound into the labyrinth We, like Arslan (1962), have begun more and more to do the greater part of the irradiation in this hollow, directed towards the ampullae and vestibule, and a decidedly lesser amount towards the lateral canal An important advantage in this is that it brings the ultrasound a greater distance from the facial nerve compared to the original technique

It would be of value to know more exactly the thickness of the bony wall, partly in order to be able to choose the best ultrasonic power and dosage and partly to be able to better direct the beam The acoustic propagation of ultrasound could be measured on the basis of absorption characteristics through the use of echo methods Investigation and development of such an instrument is under way at this time and we hope to present it at a later date

The ultrasonic dosage has been gradually increased since we found that using the present irradiation technique the higher dosages do not damage the hearing The total dosage of ultrasound given averages 2600 joules and several cases have received over 3000 joules This is a very high dosage By comparison, it can be pointed out that Angell James and co-workers have given only 650 and 1100 joules respectively to two consecutive groups of patients For the most part, we use a transducer with a diameter of 2 6 mm (surface area approx 5 mm²) and an output power of 1 5–2 watts

SUMMARY

A new Swedish ultrasonic generator, called *Ultrapoint*, has been constructed for the treatment of Meniere's disease. The principles on which this apparatus is based are as follows: high frequency alternating current is transferred from the oscillator to the transducer, in which a small barium titanate or lead zirconate disc converts the electrical energy into ultrasonic waves. The beam of rays is well concentrated with the focal point just inside or at the flat tip of the narrow, conical treatment head. Cooling is effected by means of distilled water circulating inside the transducer. The conical tips of the transducer are interchangeable and vary in diameter from 1.5 to 2.6 mm. Obliquely bevelled tips have also been produced to facilitate application in cases of difficult anatomy. The whole treatment head, including the cable, can be sterilized by boiling or autoclaving.

A new simple device for measuring the ultrasonic output of the treatment head has been designed—the so-called Intensimeter.

The radiation pattern of the new ultrasonic transducer has been studied by means of a Schlieren optical system. The relationship between the ultrasonic intensity at a given point in the radiation field and the light deflected in the Schlieren system is not a simple linear one. At relatively low sound intensities the relationship is almost linear but this soon reaches a point of saturation at which an increase in sound intensity is not followed by a proportional increase in the light output of the Schlieren system.

The ultrasonic propagation and absorption in bone has been tested on flat slices of the femoral cortex. Bone of 4 mm thickness is not penetrated, while thinner bone slices allowed the ultrasound to penetrate in direct proportion to their thinness.

Ultrasound can produce standing waves in various materials, among which, blood. This characteristic has been made use of regularly during the operations as a very simple and rapid check that the apparatus is functioning ("glass slide test").

This tendency to standing wave formation in fluid has been utilized for visualizing the ultrasonic beam in the operation cavity. The operator can direct the beam against selected targets and so see exactly at which point it enters the labyrinth. With this technique damage to the facial nerve can be avoided.

The biological effect of the new Swedish ultrasonic apparatus has been tested on pigeons. Electronystagmographical recordings of the pigeons'

head nystagmus during the rotation test have shown different degrees of functional impairment after irradiation. Morphologically have been noted varying degrees of degeneration of both the neuro-epithelium and of the secretory epithelium, damage to the cupulae, proliferative changes in the bony walls, varying degrees of obliteration of the perilymphatic space, and thickening of the walls of the membranous labyrinth

Good correlation has been shown to exist between the morphological and functional findings in the experimental animals

Fifty patients suffering from Meniere's disease have been treated up to now. The average figure for the total power used is 2600 joules and the average irradiation time has been 32 minutes The following results have been obtained. freedom from or improvement of vertigo in 45 cases, hearing improved in 6 cases, unchanged in 28 and worsened in 16, tinnitus improved in 11 and unchanged in 39 cases Facial paralysis has occurred in one case and spontaneously disappeared Post-operative symptoms of giddiness have been insignificant throughout

A careful nystagmographical analysis of the caloric test before and two months after irradiation has been made in the first 37 patients treated After irradiation the caloric excitability was eliminated in six cases, whereas in the remaining 31 cases a reduced or in some cases a normal caloric reaction could be elicited Transitory elimination of caloric excitability has been noted

An electronystagmographic record is made during the ultrasonic irradiation Homolateral or "irritative" and contralateral or "paralytic" nystagmus have been recorded

Repeated transitory changes in direction of ultrasonically induced nystagmus from the treated to the non-treated side have been recorded This is not a sign of destruction but probably an indication of temporary loss of vestibular function on the irradiated side

Our opinion is that the ultrasonic treatment of Meniere's disease in man leads to a partial extinction of the vestibular function and to a hypothetical effect on the secretory epithelium with a reduction of the endolymph secretion with a diminuation of the endolabyrinthine hydrops The latter postulate may be considered supported by our experimental studies on irradiated pigeon labyrinths

The results of most clinics have been up to now so favorable that we for the future believe that we are entitled to recommend ultrasonic irradiation in relatively early stages of the illness with a fair chance of the patient finding relief from his crises of nausea and vertigo through a selective and partial destruction of the vestibular apparatus

ZUSAMMENFASSUNG

Ein neuer schwedischer Ultraschallgenerator „Ultrapoint“ genannt, ist zur Behandlung des Morbus Menière konstruiert worden. Die Prinzipe dieser Konstruktion sind folgende:

Hochfrequenter Wechselstrom wird durch einen Oscillator zu einem Senderteil überführt, in welchem ein kleiner konkaver Geber aus Bariumtitanat oder Bleizirkonat schwingt und dabei die elektrische Energie in Ultraschallwellen verwandelt. Das Schallwellenbündel wird genau im Brennpunkt innerhalb des flachen Endes des konischen Behandlungskopfes (Schallkopfes) konzentriert. Die Kühlung des Senders erzielt man durch destilliertes Wasser, welches durch den inneren Sender zirkuliert. Die konischen Schallköpfe des Senders, deren Diameter zwischen 1,5 und 2,6 mm variiert, sind auswechselbar. Auch schräge Schallköpfe sind hergestellt worden um deren Anwendung bei Fällen mit schwieriger Anatomie zu ermöglichen.

Die gesamte Behandlungseinheit, einschliesslich dem Kabel, kann durch Kochen oder im Autoklaven sterilisiert werden.

Eine neue einfache Anordnung zum Messen der Ultraschallstärke im Schallkopf, das sogenannte „Intensimeter“, ist entwickelt worden.

Die Strahlung des neuen Ultraschallsenders ist mit Hilfe eines Schlieren-optischen Systems studiert worden. Das Verhältnis zwischen Ultraschallintensität in einem gegebenen Punkt des Strahlungsfeldes und dem abgelenkten Licht im Schlierensystem ist nicht linear. Bei anfangs geringer Schallintensität ist das Verhältnis nahezu direktproportional. Es wird jedoch bald jener Sättigungspunkt erreicht, von wo an einer Steigerung der Schallintensität keine entsprechende Lichtänderung im Schlierensystem mehr folgt.

Die Fortpflanzung und Absorption des Ultraschalls im Knochen wurde in flachen Schnitten des femoralen Cortex geprüft. Knochen von 4 mm Dicke werden nicht, dünnere dagegen werden in steigendem Masse mit abnehmender Schichtdicke durchdrungen.

Ultraschall kann in verschiedenen Stoffen, z.B. Blut, stehende Wellen bilden. Diese Eigenschaft wurde bei Operationen regelmässig als eine sehr einfache Funktionsprobe der Apparatur ausgenutzt („Standing wave test“).

Die Neigung zur Wellenbildung in Flüssigkeiten ist zum Sichtbarmachen des Strahlenbüschels im Operationsfeld benutzt worden. Der Operateur

kann den Strahl auf das gewählte Ziel richten und genau sehen, wo dieser in das Labyrinth eintritt. Mit Hilfe dieser Technik können Schäden am Facialisnerven vermieden werden.

Der biologische Effekt des neuen schwedischen Ultraschallapparates ist an Tauben geprüft worden Elektro-nystagmographische Aufzeichnungen des Taubenkopfnystagmus während des Rotationstests haben nach Bestrahlung verschieden grosse funktionelle Verminderung gezeigt Morphologisch hat man Degeneration von sowohl des Neuroepithels als auch des sekretorischen Epithels, Schaden der Cupulae, proliferative Veränderungen in den Knochenwänden, verschiedene Grade der Obliteration des perilymphatischen Raumes und Verdickung der Membranwände des Labyrinths verzeichnet.

Es ist gezeigt worden, dass eine gute Übereinstimmung zwischen den morphologischen und den funktionellen Funden an den Versuchstieren besteht

Bis jetzt sind funfzig Patienten, die an Morbus Menière litten, behandelt worden. Die durchschnittlich angewandte Totaldosis betrug 2600 Joules und die gewöhnliche Bestrahlungsdauer war 32 Minuten

Folgende Resultate wurden erhalten Befreiung von, oder Verbesserung von Schwindel in 45 Fallen Gehör Verbessert in 6, unverändert in 28, und verschlechtert in 16 Fällen. Ohrenklingen Verbessert in 11 und unverändert in 39 Fällen

Facialisparese ist in einem einzigen Fall vorgekommen und spontan zuruckgegangen Die postoperativen Schwindelsymptome sind durchwegs unbedeutend gewesen

Es wurde eine sorgfaltige nystagmographische Analyse des kalorischen Tests vor und 2 Monate nach Bestrahlung der ersten 37 behandelten Patienten ausgefuhrt

Nach Bestrahlung war bei sechs Patienten die kalorische Reizbarkeit ausgelöscht, in den verbleibenden 31 Fällen stark vermindert, oder bei einigen Patienten normal

Temporärer Fortfall der kalorischen Reizbarkeit ist beobachtet worden

Wahrend der Bestrahlung wurde Elektronystagmographie ausgefuhrt Homolateraler oder „irritativer" und kontralateraler oder „paralytischer" Nystagmus wurden registriert

Wiederholte transitorische Veränderungen in der Richtung des Nystagmus, von homolateral nach kontralateral wurden beobachtet Dies ist kein Beweis fur Destruktion, aber möglicherweise ein Zeichen fur zeitlichen Verlust der vestibularen Funktion auf der beschallten Seite

Wir sind der Meinung, dass die Ultraschallbehandlung am Menschen, mit unserer Methode, in den meisten Fällen zu einer partiellen Destruktion der vestibulären Funktion und möglicherweise einer Verminderung der Endolymphatischen Sekretion fuhrt Die letztere Behauptung wird durch unsere Studien am bestrahlten Taubenlabyrinth entschieden unterstutzt

Bis heute sind die meisten klinischen Resultate so erfreulich gewesen, dass wir uns in der Zukunft berechtigt fühlen die Ultraschallbehandlung schon in einem relativ frühen Stadium der Krankheit rekommandieren zu können. Dabei besteht für den Patienten gute Aussicht, durch eine selektive und partielle Destruktion des vestibulären Apparates von seinen Nausea- und Vertigobeschwerden befreit zu werden.

RÉSUMÉ

Un nouveau générateur d'ultra-sons suédois, appelé l'Ultrapoint et destiné au traitement de la maladie de Menière, a été construit. Les principes sur lesquels se base le fonctionnement de cet appareil sont les suivants. — Un courant alternatif à haute fréquence est transféré de l'oscillateur au transducteur, dans lequel un petit disque de titanate de barium ou de zirconate de plomb transforme l'énergie électrique en ondes ultrasoniques. Le faisceau de rayons est bien concentré, le foyer se trouvant à l'extrémité platte de la tête de traitement ou immédiatement en dedans de cette extrémité. Le refroidissement s'effectue à l'aide d'eau distillée circulant à l'intérieur du transducteur. Les pointes coniques du transducteur sont interchangeables et leurs diamètres varient de 1,5 à 2,6 mm. Des pointes à extrémité oblique ont aussi été produites pour faciliter l'application en cas d'anatomie difficile. L'ensemble de la tête de traitement, y compris le câble, peut être stérilisé par ébullition ou en autoclave.

Un nouveau moyen de mesurer l'intensité ultrasonique a été mis au point — l'Intensimètre.

La conformation des radiations émises par ce nouveau transducteur ultrasonique a été étudiée à l'aide d'un système optique Schlieren. La relation entre l'intensité à un point donné du champ de radiation et la lumière déviée dans le système Schlieren n'est pas simplement linéaire. A des intensités sonores relativement basses, la relation est presque linéaire, mais ceci atteint bientôt un point de saturation où une augmentation de l'intensité sonore n'est plus accompagnée d'une augmentation proportionnelle de la production de lumière du système Schlieren.

La propagation et l'absorption des ultra-sons dans le tissu osseux a été étudiée sur des tranches plattes de l'écorce du fémur. Une épaisseur de 1 mm d'os n'est pas pénétrée, mais des tranches d'os plus minces laissent pénétrer les ultra sons en proportion directe de leur minceur.

Les ultra-sons peuvent produire des ondes stationnaires dans certains matériaux, entre autres le sang. Ce phénomène a été régulièrement employé pendant les opérations pour contrôler d'une façon simple et rapide que l'appareil fonctionne (« standing wave test »).

Cette tendance à former des ondes stationnaires dans les liquides a été utilisée pour rendre visible le faisceau ultrasonique dans la cavité opératoire. L'opérateur peut diriger le faisceau vers des cibles choisies et peut ainsi voir exactement en quel point il pénètre dans le labyrinthe. Grâce à cette méthode on peut éviter de léser le nerf facial.

Les effets biologiques du nouvel appareil ultrasonique suédois ont été

études sur le pigeon. L'enregistrement électronystagmographique du nystagmus de la tête du pigeon pendant la rotation a indiqué un degré variable de détérioration après l'irradiation. Du point de vue morphologique on a pu constater un degré variable de dégénération du neuroépithélium et de l'épithélium sécrétoire, des lésions des coupoles, des altérations prolifératives des cloisons osseuses, un degré variable d'oblitération de l'espace périlymphatique et un épaississement des parois du labyrinthe membraneux.

On a constaté une bonne corrélation entre les observations morphologiques et fonctionnelles chez les animaux.

Jusqu'ici 90 malades souffrant de la maladie de Ménière ont été traités. La valeur moyenne de la puissance totale employée est de 2600 Joules et la durée moyenne de l'irradiation de 32 minutes. On a obtenu les résultats suivants: disparition ou amélioration du vertige dans 45 cas; amélioration de l'audition dans 6 cas, audition inchangée dans 28 cas et empirée dans 16 cas; bourdonnements d'oreilles améliorés dans 11 cas et inchangés dans 39 cas. Une paralysie faciale s'est déclarée dans un cas et a disparu spontanément. Les symptômes post-opératoires d'étourdissement ont toujours été insignifiants.

Une analyse nystagmographique soigneuse de l'épreuve thermique avant l'irradiation et deux mois après celle-ci a été exécutée sur les 37 premiers malades traités. Après l'irradiation l'excitabilité thermique avait été éliminée dans 6 cas et dans les 31 autres cas une réaction thermique réduite ou, dans peu de cas, normale pouvait être produite. Une diminution passagère de l'excitabilité thermique a été notée.

L'enregistrement électronystagmographique se fait pendant l'irradiation ultrasonique. On a enregistré un nystagmus homolatéral (ou « irritatif ») et un nystagmus contralatéral (ou « paralytique »).

Des changements répétés de la direction du nystagmus produit par les ultra-sons, du nystagmus homolatéral au nystagmus contralatéral, ont été constatés. Ceci n'est pas un signe de destruction, mais probablement une indication d'une perte temporaire de la fonction du vestibule du côté irradié.

Selon notre opinion le traitement ultrasonique du syndrome de Ménière chez l'homme donne lieu à une extinction partielle de la fonction vestibulaire et à un effet hypothétique sur l'épithélium sécrétoire avec une réduction de la sécrétion endolymphatique et une diminution de l'hydrops endolabyrinthique. Ce dernier postulat peut être considéré confirmé par nos recherches expérimentales sur les labyrinthes de pigeon irradiés.

Les résultats de la plupart des cliniques ont été si favorables jusqu'ici que nous estimons être autorisés de recommander à l'avenir l'irradiation ultrasonique dans des phases relativement récentes de la maladie avec de bonnes chances pour le patient de trouver un soulagement de sa crise de nausée et de vertige par une destruction sélective et partielle de l'appareil vestibulaire.

REFERENCES

ALTMANN F., 1962 Experiences with the ultrasonic treatment of Meniere s disease at the Presbyterian Hospital in New York International Symposium on Meniere s disease Padua Sept 11th and 12th

ALTMANN F., and WALTNER J G., 1961 Treatment of Menière s disease with ultrasonic waves *Arch Otolaryng* 74 27

—— 1961 The present status of the treatment of Meniere s disease with ultrasound *Bull N Y Acad Med* 37, 621

ANDERSON, T O., WAKIM, K G., HERRICK J F., BENNETT, W A., and KRUSEN F H., 1951 An experimental study of the effect of ultrasonic energy on the lower part of the spinal cord and peripheral cord nerves *Arch phys Med* 32 71

ANGELL JAMES, J., DALTON G A., BULLEN M A., FREUNDLICH H F., and HOPKINS, J C., 1960 The ultrasonic treatment of Meniere s disease *J Laryng* 74 730

ANGELL JAMES, J., DALTON G A., BULLEN M A., FREUNDLICH H F., and WELLS, P N T., 1961 The effect of ultrasonics on the temporal bone *Acta oto laryng (Stockh)* 53, 168

ANGELL JAMES J., DALTON G A., HADLEY K J., FREUNDLICH H F., BULLEN M A., and WELLS P N T., 1963 A new 3 megacycle generator for destruction of the vestibular end organ *Acta oto laryng (Stockh)* (in press)

ARDEN, N I., JR., JANES J M., and HERRICK J F., 1957 Ultrasonic energy and surgically produced defects in bone *J Bone Jt Surg* 39 A 394

ARIAGNO R P., 1960 Treatment of Meniere s disease with ultrasound *Arch Otolaryng* 71 513

—— 1962 Four years of ultrasound in Meniere s disease *Arch Otolaryng* 76 30

ARSLAN M., 1953 *Treatment of Menière s syndrome by direct application of ultra sound waves to the vestibular system* Proc Vth Int Congr Otorhinolar Amsterdam

—— 1955 Applikation des Ultraschalles auf das Labyrinth Ein Beitrag zur Therapie der Labyrinthose *Arch Ohr Nas u Kehlk Heilk* 167 559

—— 1958 Ultrasonic surgery of the labyrinth in patients with Meniere s syndrome *Sci med ital* VII 301

—— 1960 Die Zerstörung des Vestibularapparates durch Ultraschall bei der Menierschen Krankheit *Dtsch med Wschr* 85 1573

—— 1962 An improved technique of the ultrasonic irradiation of the vestibular apparatus by Meniere s disease *Acta oto-laryng (Stockh)* 55 461

ASCHAN G., 1955 The mechanism of the cupula ampullaris in man *Acta Soc Med upsalien* 60 77

ASCHAN G., BERGSTEDT M., and STAHLE, J 1955 Nystagmographical observations illustrating the cupular mechanism in rabbits and pigeons *Acta Soc Med upsalien* 60 89

—— 1956 Nystagmography Recording of nystagmus in clinical neuro-otological examinations *Acta oto laryng (Stockh* Suppl 129

BALDES, J F., 1958 Biologic effects of ultrasound *Amer J phys Med* 37 11

BALLANTINE, JR H T HUETER, T F., NAUTA W J H., and SOSA D M., 1956 Focal destruction of nervous tissue by focussed ultrasound Biophysical factors influencing its application *J exp Med.*, 104 337

BARNARD, J. W., FRY, W. J., FRY, F. J., and BRENNAN, J. F., 1956. Small localized ultrasonic lesions in the white and gray matter of the cat brain. *Arch. Neurol. Psychiat. (Chic.)* 75: 15.

BARNARD, J. W., FRY, W. J., FRY, F. J., and KRUMINS, R. F., 1955. Effects of high intensity ultrasound on the central nervous system of the cat. *J. comp. Neurol.*, *103*: 459.

BARTH, G., and BÜLOW, H. A., 1949. Zur Frage der Ultraschallschädigung jugendlicher Knochen. *Strahlentherapie 79*, 2.

BASAURI, L., and LELE, P. P., 1962. A simple method for production of trackless focal lesions with focussed ultrasound. Statistical evaluation of the effects of irradiation of the central nervous system of the cat. *J. Physiol. (Lond.) 160*: 513.

BECK, CHL., 1958. Ultraschallwirkung am menschlichen Bogengang. *Arch. Ohr.-, Nas.-, u. Kehlk. Heilk. 172*, 513.

—— 1959. Die Antwort des Cortischen Organs auf transkapsuläre Ultraschallapplikation (Experimentelle Untersuchungen am Meerschweinchen). *Arch. Ohr.-, Nas.-, u. Kehlk. Heilk. 174*: 173.

BENDER, L. F., JANES, J. M., and HERRICK, J. F., 1954. Histologic studies following exposure of bone through ultrasound. *Arch. phys. Med. 35*: 555.

BERGGREN, E., 1939. Contribution toward the surgical treatment of morbus Menière. *Acta oto-laryng. (Stockh.) 27*, 636.

BERGGREN, S., 1949. Histological investigation of three cases with Menière's syndrome. *Acta oto-laryng. (Stockh.)*, *37*: 30.

BERGMANN, L., 1949. Der Ultraschall und seine Anwendung in Wissenschaft und Technik. S. Hirzel Verlag Zurich.

BRAIN, D. J., COLMAN, B. H., LUMSDEN, R. B., and OGILVIE, R. F., 1960. The effects of ultrasound on the internal ear. A histological investigation. *J. Laryng. 74*: 628.

BRUNNER, H., 1925. Die Pathologie und Therapie der vasomotorischen Erkrankungen des Innenohres. *Wien. klin. Wschr. 38*: 1235.

BUCHTALA, V., 1948. Die Ultraschallwirkung auf den wachsenden Knochen. *Strahlentherapie 78*: 127.

BULLEN, M. A., WELLS, P. N. T., FREUNDLICH, H. A., and ANGELL JAMES, J., 1963. A physical survey of the ultrasonic treatment of Menière's disease. *Ultrasonics 1*: 2.

CAWTHORNE, T. E., 1943. The treatment of Menière's disease. *J. Laryng. 58*: 363.

—— 1947. Menière's disease. *Ann. Otol. (St. Louis) 56*: 18.

—— 1957. Membranous labyrinthectomy via the oval window for Menière's disease. *J. Laryng. 71*: 524.

—— 1960. Labyrinthectomy. *Ann. Otol. (St. Louis) 69*: 1170.

—— 1962. Lecture given at the meeting of the Danish Otolaryngological Society April 6.

CAWTHORNE, T., and HEWLETT, A. B., 1954. Menière's disease. Proc. roy. Soc. Med. 47: 663.

CERNY, E., HOLLUB, M., and TUREK, J., 1962. Treatment of Menière's disease with ultrasound. International Symposium on Menière's disease. Padua Sept. 11th and 12th.

CRAWFORD, A. E., 1955. Ultrasonic engineering. Butterworths Scientific Publications London.

DANDY, W. E., 1933. Menière's disease. Diagnosis and treatment. *Amer. J. Surg. 20*: 693.

—— 1941. The surgical treatment of Menière's disease. *Surg. Gynec. Obstet. 72*: 421.

DAY, K. M., 1946. Hydrops of labyrinth (Menière's disease). Diagnosis. Results of labyrinth surgery. *Laryngoscope (St. Louis) 56*: 33.

DEDERDING, D., 1931. Our Menière treatment (principles and results). *Acta oto-laryng. (Stockh.) 16*: 404.

DIX, M. R., and HALLPIKE, C. S., 1952. The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system. *Proc. roy. Soc. Med. 45*: 341.

DOGNON, A., and BIANCANI, E. H., 1937. Ultrasons et biologie. Gauthier-Villars Paris.

DOHLMAN, G., 1959. Modern views on vestibular physiology. *J. Laryng. 73*: 154.

Dibs, R., 1957 Über die Ultraschallbehandlung des Morbus Menière nach der Methode von Arslan *Pract oto rhino laryng (Basel), 19* 401

Dussik, K., 1942 Über die Möglichkeit hochfrequente mechanische Schwingungen als diagnostisches Hilfsmittel zu verwenden *Z Neurol 174* 153

Edler I., 1961 Ultrasound cardiography *Acta med scand., Suppl 370*

Ewald J R., 1892 Physiologische Untersuchungen über das Endorgan des Nervus Octavus

Fitzgerald G., and Hallpike, C. S., 1942 Studies in human vestibular function I Observations on the directional preponderance of caloric nystagmus resulting from cerebral lesions *Brain 65* 115

Frenckner, P., 1952 Some view points on the operative treatment in Menière's disease *Arch Otolaryng 55* 420

Fry F J., 1958 Precision high intensity focusing ultrasonic machines for surgery *Amer J phys Med 37*, 152

—— 1953 Action of ultrasound on nerve tissue—a review *J acoust Soc Amer 25* 1

—— 1954 Intense ultrasound—a new tool for neurological research *J ment Sci 100* 85

—— 1958 Use of intense ultrasound in neurological research *Amer J phys Med 37* 143

—— 1958 Intense ultrasound in investigations of the central nervous system *Advanc biol med Phys VI* 281

—— 1962 Present and future applications of ultrasonics in biomedicine *Proceedings of the IRE 50* 1393

Fry W J., and Fry F J., 1960 Fundamental neurological research and human neurosurgery using intense ultrasound *IRE transactions on medical electronics MF 7* 166

Fry W J., and Fry R B., 1953 Temperature changes produced in tissue during ultrasonic irradiation *J acoust Soc Amer 25* 6

Fry W J., Barnard J W., Fry F J., and Brennan J F., 1955 *Ultrasonically produced localized selective lesions in the central nervous system Amer J phys Med 34* 413

Fry W J., Barnard J W., Fry F J., Krumins R F., and Brennan J F., 1955 Ultrasonic lesions in the mammalian central nervous system *Science 122* 517

Galambos R., 1942 The avoidance of obstacles by flying bats Spallanzani's ideas (1794) and later theories *Isis (Seattle) 34* 132

Gordon D., 1960 The performance of ultrasonic transducers for the surgery of the labyrinth Third international conference on medical electronics 26th July

Grabar P., and Prudhomme, P O., 1949 Die mechanischen und die chemischen Wirkungen der Ultraschallwellen Kongressbericht der Erlanger Ultraschalltagung p 114 S Hirzel Verlag Zurich

Griffin D R., 1944 How bats guide their flight by supersonic echoes *Amer J Physics 12* 342

Hallpike C S., and Cairns, H., 1938 Observations on the pathology of Menière's disease *J Laryng 53* 625

Harvey E N., and Loomis, A L., 1931 High speed photomicrography of living cells subjected to the supersonic vibrations *J gen Physiol 15* 147

Hasse, C., 1867 Der Bogenapparat der Vögel *Z wiss Zool 17* 598

Heiberts, G., Rydmark K E., and Stahle J., 1953 1954 Partiell labyrintdestruktion med galvanokaustik *Svensk otolaryngol foreningsförhandl 20*

Herrick J F 1953 Temperatures produced in tissues by ultrasound Experimental study using various technics *J acoust Soc Amer 25* 12

Ironside, W M S and Lindsay J R., 1959 Ultrasonic therapy for relief of vertigo due to Menière's disease *Laryngoscope (St Louis) 69* 833

JANES, J M., KELLY, P J., HERRICK, J F., and PETERSON, L F A., 1962 The effect of high-dosage ultrasonic energy on femura of the dogs *J Bone Jt Surg*, 44 A, 1293

JEPPSON S., 1961 Echo encephalography IV The midline echo An evaluation of its usefulness for diagnosing intracranial expansivities and an investigation into its sources *Acta chir scand*, Suppl 272

JONGKEES, L B W., MAAS, J P M., and PHILIPSZOON, A J., 1962 Clinical nystagmography *Pract oto rhino laryng (Basel)*, 24, 65

JURINE, L., 1798 Cit BERGMANN, L Der Ultraschall, 1954 p 674 Hirzel Verlag, Stuttgart

KREJCI, F., 1951 Statistische Untersuchungen uber den Wert der Ultraschalltherapie bei Ohrenkrankungen *Wien klin Wschr* 63 707

—— 1952 Experimentelle Grundlagen einer extralabyrinthären chirurgischen Behandlungsmethode der Menierschen Erkrankung *Pract oto rhino laryng (Basel)*, 14 18

KRISTENSEN, H K., 1961 Histopathology in Menière s disease *Acta oto laryng (Stockh)*, 53 237

KUNDT, A., 1866 Uber eine neue Art akustischer Staubfiguren und uber eine Anwendung derselben zur Bestimmung der Schallgeschwindigkeit in festen Korpern und Gasen *Pogg Ann Phys*., *127*, 497 (Cit BERGMANN, 1949)

LAKE, R., 1904 Removal of the semicircular canals in a case of unilateral aural vertigo *Lancet*, *1*, 1567

LANGEVIN, M P., 1926 Sondeur ultra sonore *Recherches et Inventions BI 7*, 119

LEKSELL, L., 1955/56 Echo-encephalography I Detection of intracranial complications following head injury *Acta chir scand*, *110*, 301

—— 1957 Gezielte Hirnoperationen Handbuch der Neurochirurgie, *Bd 6*, 178 Springer Verlag, Berlin Gottingen, Heidelberg

LELE, P P., 1962 The effects of focussed ultrasonic radiation on the peripheral nerve with observations on effects of local heating Unpublished manuscript Medical acoustic research group Department of neurosurgery Massachusetts general hospital Boston 14 U S A

LIDVALL, H F., 1962 Specific and non specific traits of habituation in nystagmus responses to caloric stimuli *Acta oto laryng (Stockh)* 55 315

LINDAHL, J W S., and ROBERTSON M S., 1962 The use of ultrasound in the treatment of aural vertigo *J Laryng*, *76* 239

LINDSAY J R., 1946 Labyrinthine dropsy *Laryngoscope (St Louis)* 56 325

—— 1949 Labyrinthine surgery for Menière s disease *Laryngoscope (St Louis)*, 55 22

LINDSTROM, P A., 1954 Prefrontal ultrasonic irradiation—a substitution for lobotomy *Arch Neurol Psychiat (Chic)* 72 399

LITHANDER B., 1961 Clinical and experimental studies in echo encephalography *Acta psychiat scand* Suppl 155

LUMSDEN R B., 1958 Treatment of Menière s disease with ultrasound *Proc roy Soc Med* 51 617

MADSEN P W., and GERSTEN J W., 1961 The effect of ultrasound on conduction velocity of peripheral nerve *Arch phys Med Sept* 645

McLAY K., FLINN M., and ORMEROD F C., 1961 Histological changes in the inner ear resulting from the application of ultrasonic energy *J Laryng* 75 345

MAJNO G., 1949 Knochen und Sehnenlasionen durch Ultraschall Kongressbericht der Erlanger Ultraschalltagung p 205 S Hirzel Verlag Zurich

MARINESCO N., 1932 Action du piezo-quartz oscillant sur les sols et les suspensions *C R Acad Sci (Paris)* 194 1824

MATTHES, K., and RECH W., 1949 Der Ultraschall in der Medizin Kongressbericht der Erlanger Ultraschalltagung S Hirzel Verlag Zurich

MEURMAN Y., 1951 Einwirkung einfacher Fenestration auf die Menièrsche Krankheit *Arch Ohr Nas u Kehlk Heilk* 157 439

MILLIGAN W., 1904 Menière s disease A clinical and experimental inquiry *Brit med J* 2 1228

MOLLISON, W. M., 1935. Operative treatment of vertigo. *Proc. roy. Soc. Med.* 28, 1597.

—— 1939. Surgical treatment of vertigo by opening the external semicircular canal and injecting alcohol. *Acta oto-laryng. (Stockh.)*, 27, 245.

MEYERS, R., FRY, W. J., FRY, F. J., DREYER, L. L., SCHULTZ, D. F., and NOYES, R. F., 1959. Early experiences with ultrasonic irradiation of the pallidofugal and nigral complexes in hyperkinetic and hypertonic disorders. *J. Neurosurg.* 16, 32.

MYGIND, S. H., 1938. Clinical experiments with reference to the influence of water metabolism on the ear. *Ann. Otol. (St. Louis)* 47, 360.

MYGIND, S. H., and DEDERDING, D., 1932. The significance of water metabolism in general pathology as demonstrated by experiments on the ear. *Acta oto-laryng. (Stockh.)*, 17, 424.

MULWERT, H., and VOSS, O., 1928. Eine neue physikalische Behandlungsmethode chronischer Schwerhorigkeit und deren Ergebnisse. *Acta oto-laryng. (Stockh.)* 12, 63.

OLIVECRONA, H., 1943. Om Menières sjukdom och dess kirurgiska behandling. *Nord. Med.* 19, 1463.

ORMEROD, F. C., 1960. The physiology of the endolymph. *J. Laryng.* 74, 659.

PETERS, G., 1949. Ultraschallwirkung auf das Nervensystem. Kongressbericht der Erlanger Ultraschalltagung, p. 166. S. Hirzel Verlag, Zurich.

POHLMAN, R., 1939. Über die Ausbreitung und Absorption von Ultraschall im menschlichen Gewebe und seine therapeutische Wirkung bei Ischias und Plexusneuralgie. *Verh. deutsche phys. Ges. (3)* 20, 52.

—— 1951. Die Ultraschalltherapie. Huber, Bern.

PORTMANN, G., 1927. The saccus endolymphaticus and an operation for draining the same for the relief of vertigo. *J. Laryng.* 42, 809.

PORTMANN, G., PORTMANN, M., and BARBE, L. J., 1951. Étude expérimentale (Fonctionelle et histologique) de l'action des ultra sons sur l'audition. *Acta oto-laryng. (Stockh.) Suppl.* 100, 113.

RETZIUS, G., 1884. Das Gehororgan der Wirbeltiere II. Stockholm.

ROLLIN, H., 1940. Zur Kenntnis des Labyrinthhydrops und des durch ihn bedingten Menière. *Hals- Nas- u. Ohrenar-t (1 Teil)* 31, 73.

ROSSBERG, G., 1954. Drehschwindel und Spontannystagmus. *Arch. Ohr.- Nas.- u. Kehlk.-Heilk.* 165, 417.

SAXEN, A., 1948. Histologische Untersuchungen uber die Sekretion und Resorption der Endolymphe im Innenohr. *Bull. Schweiz. Akad. med. Wiss.* 3, 250.

SCHMID, G., 1949. Abbau von Makromolekeln durch Ultraschall. Kongressbericht der Erlanger Ultraschalltagung, p. 55. S. Hirzel Verlag, Zurich.

SCHUKNECHT, H. F., 1957. Ablation therapy in the management of Menière's disease. *Acta oto-laryng. (Stockh.) Suppl.* 132.

SEIDL, F., and KREJCI, F., 1952. Untersuchungen uber die Durchdringbarkeit des Schlafenbeins mit Ultraschall mittels der Schlierenmethode. *Pract. oto-rhino-laryng.* 14, 65.

SPALLANZANI, L., 1793. Cit. BERGMANN, L. Der Ultraschall, p. 674. Hirzel Verlag, Stuttgart.

STAHLE, J., 1954–1955. Fluorens-Ewalds lag. *Svensk otolaryng. forenings forhandl.* 20.

—— 1956. Electronystagmography in the caloric test. *Acta Soc. Med. upsalien.* 61, 307.

—— 1957. Electronystagmography in the rotatory test. *Acta Soc. Med. upsalien.* 62, 77.

—— 1958. Electro-nystagmography in the caloric and rotatory tests. A clinical study. *Acta oto-laryng. (Stockh.) Suppl.* 13.

—— 1961. Discussion at the symposium oto-neurologicum, Basel, 1960. *Acta oto-laryng. (Stockh.) Suppl.* 1?9, 120.

DE STEFANI, G. B., 1956. Gli ultrasuoni nell'otosclerosi. *Arch. ital. Otol. Suppl.* 2?.

STRAUBEL, H., 1931. Einige Versuche mit Ultraschall. *Physikalische Zeitschrift* 32, 379.

VAN WENT, J., 1954. Ultrasonic and ultrashort waves in medicine. Elsevier Publishing Company, Amsterdam, Houston, London, New York.

WIETHE, C., 1949. Die Behandlung des Menière mit Ultraschall. *Wien. klin. Wschr.* 61, 466.

WIETHE, C., WYT, L., and YSSLONZIL, I., 1948 The therapeutic use of supersonic waves *Acta oto-laryng (Stockh.)*, *Suppl 78* 111

WITTMAACK, K., 1926 Die entzündlichen Erkrankungsprozesse des Gehororgans In Handbuch der spez path Anat u Hist Hgb v F Henke u O Lubarsch *12* 102 Springer Verlag, Berlin

—— 1956 Die Ortho- und Pathobiologie des Labyrinthes George Thieme Verlag, Stuttgart

WRIGHT, A J., 1938 Labyrinthine destruction in the treatment of vertigo by injection of alcohol through the oval window *J Laryng*, *53* 504

WUSTROW, F., and BORKOWSKY, B., 1960 Ergebnisse nach konservativen und chirurgischen Behandlungsmethoden sowie kritische Betrachtungen zur Pathogenese des Morbus Menière *Z Laryng Rhinol* *39*, 133

YSSLONZIL, I., 1949 Über die Beeinflussung des Vestibularapparates durch Ultraschall *Wien klin Wschr* *61*, 468

YOUNG, R R., and HENNEMAN, E., 1961 Functional effects of focussed ultrasound on mammalian nerves *Science*, *134*, 1521

ÅSTRÖM, K E., BELL, E., BALLANTINE, JR., H T., and HEIDENSLEBEN E., 1961 An experimental neuropathological study of the effects of high frequency focussed ultrasound on the brain of the cat *J Neuropath exp Neurol*, *20* 484

*

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta

OTO-LARYNGOLOGICA

S U P P L E M E N T U M 179

CONTRIBUTION TO THE VESTIBULAR PHYSIOLOGY AND VESTIBULAR TEST

BY

JAPAN SOCIETY OF VESTIBULAR RESEARCH

ACTA OTO-LARYNGOLOGICA KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO LARYNGOLOGICA

SUPPLEMENTUM 1 9

CONTRIBUTION TO THE VESTIBULAR PHYSIOLOGY AND VESTIBULAR TEST

By

JAPAN SOCIETY OF VESTIBULAR RESEARCH

TOKYO 1963

PRINTED IN JAPAN BY
Choyokai Printing Bureau
TOKYO 1963

FOREWORD

Articles presented in this supplement are the results of research work done by some members of the Japan Society of Vestibular Research during the years 1959 and 1960

The Japan Society of Vestibular Research (J S V R) was established in 1956 with the purpose of discussing thoroughly vestibular problems It consists of about 150 members as of 1962 and holds a biannual meeting

Members of the J S V R cooperated in the past two years in establishing the standard of the test methods which are accurate in detecting abnormality of the equilibratory function and easy for practice to carry out The results obtained are not yet fully published but the articles published here represent the fundamental part of this cooperative research

It is unfortunate that the long history of vestibular research in Japan has not been properly evaluated by colleagues of the world This is mainly because almost all the research work done in Japan has been published in the Japanese language Introduction of the history of vestibular research in Japan is not our purpose at this point but the J S V R can not fail to mention two of our eminent pioneers Prof Ino Kubo and Prof Teiji Hoshino

Prof I Kubo, Department of Otolaryngology of Kyushu University (1908–1930) was among the first investigators of vestibular problems in Japan although his work on caloric nystagmus was mainly performed in Germany Prof T Hoshino and his coworkers, Department of Otolaryngology of Kyoto University (1924–1945), did excellent work in each part His thought of research is still alive in a number of the present members of the Japan Society of Vestibular Research

November 1962, Niigata

Masanori Morimoto, M D
Professor and Head
Department of Otolaryngology
Niigata University

ACKNOWLEDGEMENTS

We should like to express our appreciation to Prof I Kirikae, Prof T Fukuda, Dr K Owada, Dr I Watanabe and Dr G Totsuka for arranging publication of this supplement Dr K Owada spent many hours in editorial work Without his efforts this publication would not have been realized

Some investigations mentioned in these articles were supported in part by a grant in aid from the Science Research Fund of The Japanese Ministry of Education to which we convey our thanks

CONTENTS

THE OTOLITHIC REACTION ON NYSTAGMUS BY CALORIC STIMULATIONS

KENJIRO OWADA and KIYOKO OKUBO
Tokyo, Japan

From the Department of Otorhinolaryngology (Head Prof Suzuki, M D), Keio University, School of Medicine, Tokyo

Experiments with rabbits suggested that the otolithic organ does not produce nystagmus by external stimulation alone, but they maintain certain regulatory function on the nystagmus Head movement and caloric stimulations were applied to the animals in which spontaneous nystagmus was induced by resecting the ampullar nerve. Nystagmographic recordings of these animals demonstrated changes in frequency of the nystagmus indicating reaction of the otolithic organ It was observed that the utricle and saccule as suppressive mechanism on the nystagmus in a different manner The mode of regulatory function of the utricle and the saccule on the nystagmus to one direction is antagonistic in nature

INTRODUCTION

Experimental studies by Versteegh (1927), Ulrich (1935) and Sullivan (1957) indicated that the otolithic organ itself does not produce nystagmus Jongkees (1950) observed positional nystagmus after destruction of the saccule in rabbits Dix and Hallpike (1952) reported that degenerative changes were found in the otolithic macula in a patient who had had positional nystagmus

In our previous papers (1960), it was reported that the nystagmus jerks were either increased or decreased according to the change of endolymphatic pressure on the otolithic macula

From these observations it indicates that the otolithic organ may have some influence on the appearance of caloric nystagmus

METHOD

The principle of this experiment was to observe the change of nystagmus jerks by caloric stimulations before and after the section of the utricular nerve and/or the saccular nerve Healthy rabbits were used The method of the nerve section was the same as that of described in our previous papers (1960) Exposed surface of the labyrinthine capsule was covered with a vinyl sheet in order to avoid injury to the labyrinth by irrigating water

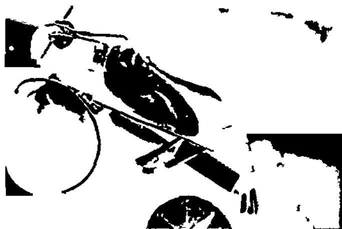


Fig 1

The first experiment was aimed to elucidate the relationship between the lymphflow during caloric stimulation and the change of pressure on the otolithic macula A rabbit was fixed on a seesaw (Fig 1) and spontaneous nystagmus was induced in this rabbit by sectioning the horizontal ampullar nerve Changes in nystagmus of rabbits with intact utricular nerve as well as the one with resected utricular nerve were observed during upward and downward motion of the head The nystagmus was recorded electronystagmographically and movements of the head was simultaneously traced by means of a photo transistor In these experiments the head movement is aimed to induce the change of pressure on the utricular macula, that is upward motion of the head is equal to the increasing pressure on the macula and vice versa

Caloric stimulations were applied with head up position before and after the section of the horizontal ampullar nerve, utricular and saccular nerve Hot and cold water was irrigated continuously into the ear canal Nystagmus jerks were counted for 30 seconds of duration starting two minutes after the beginning of irrigation



Fig 2 Resected horizontal ampullar nerve

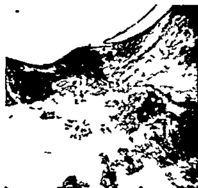


Fig 3 Resected horizontal ampullar and utricular nerve

The condition of the sectioned nerves were examined by microscopic examination after the experiment (Fig 2 and 3)

RESULTS

The results of these experiments are shown in Fig 4 and 5

In Fig 4, the upper line shows nystagmus jerks after section of the horizontal ampullar nerve The lower line indicates the movement of the head, this also represents the change of pressure applied on the utricular macula, in which the ascending line indicates decreasing pressure and the descending line corresponds to the increasing pressure When the utricular nerve was intact, the interval of nystagmus was altered according to the movement of the head (Fig 4), however after the resection of the *utricular nerve no change of nystagmus was observed* (Fig 5)



Fig 4 Utricular nerve intact upper line indicates nystagmus jerks, lower line indicates the movements of the head Horizontal line indicates still position of the head The ascending portion of the curve indicates downward motion of the head (decreasing otolithic pressure against the macula) Descending line indicates upward motion of the head (increasing pressure against the macula)

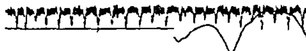


Fig 5 Utricular nerve resected upper line indicates nystagmus Lower line the head movement. Left half of the diagram shows nystagmus rhythm during head still position Right half shows unchanged nystagmus rhythm during the head movement

In order to ascertain the influence of the utricle upon nystagmus, the second experiments were carried out and following results were obtained

When the horizontal ampullar nerve of one ear was resected, spontaneous nystagmus toward the opposite side appeared In rabbits with intact otolithic organ, this nystagmus was suppresed by cold caloric stimulation to the contralateral normal ear However, when the utricular nerve was sectioned in addition to the ampular nerve, stronger cold caloric

stimulation to the other ear was necessary to suppress the nystagmus This phenomenon was not observed in rabbits with saccular nerve section

Table I *Change of nystagmus jerks induced by section of various nerves during cold caloric stimulations to the contralateral normal ear*

Resected nerve	Case No	Spontaneous nystagmus	Change of spontaneous nystagmus	Temperature of irrigated water
Horizontal ampullar nerve	43	150	0	18°C
Horizontal ampullar nerve and utricular nerve	16 18	67 150	11 23	6°C 8°C
Horizontal ampullar nerve and saccular nerve	29	150	0	24°C
VIII Nerve	51	120	8	4°C

Table II *Change of nystagmus jerks after cold(10°C) and hot(50°C) caloric stimulation into the ear which received nerve section of various combination. Number indicates the count of jerks in 30 seconds period during calorization*

Group	Resected nerve	Spontaneous nystagmus	Cold	Hot
I	Horizontal ampullar nerve	150	100	230
II	Horizontal ampullar nerve and utricular nerve	65	100	31
III	Horizontal ampullar nerve and saccular nerve	150	130	155
IV	Horizontal ampullar nerve utricular nerve and saccular nerve	150	150	120

Changes of spontaneous nystagmus by hot (50°C) and cold (10°C) caloric stimulation to the operated ear of rabbits with various combination of the nerve section are tabulated in Table II

In this table it was notable that the response of caloric stimulation in the ear with utricular nerve section is reverse to the response of the ear with saccular nerve section

DISCUSSION

The relationship between the otolithic organ and nystagmus has been studied by Versteegh, Sullivan and others Versteegh (1927) stated that no nystagmus was elicited either by destruction of the otolithic organ or by resection of the utricular nerve Sullivan (1959) observed deviation of eyes in cats after resecting the utricular nerve, but he reported no ny-

stagmus Jongkees (1950) reported the results of caloric stimulation of the labyrinth in rabbits after destruction of the saccular nerve In his experiments no change in the duration of nystagmus was mentioned

In the present experiments when the utricle was stimulated by means of up and down motion of the head in the presence of a spontaneous nystagmus, a change of nystagmus jerks was observed (Fig 4)

It was found that, when the utricle was stimulated by pressure of the otolith against the utricular macula (upward motion of the head) the spontaneous nystagmus became less remarkable However when the utricular nerve was resected no change in the interval of nystagmus to the other side was observed during the head motion (Fig 5) Likewise after the utricular nerve section of one ear the spontaneous nystagmus became difficult to be suppressed by cold caloric stimulation to the other ear These observation suggested that the utricle acts as inhibitory mechanism when the utricular macula is pressed

In order to explain the results of caloric nystagmus in Table I and II, mechanism of three receptors in the labyrinthine vestibule are to be considered They are, the crista ampularis the utricle and the saccule Under ordinary condition, all three receptors will respond to caloric stimulations However, when the horizontal ampullar nerve and the utricular nerve are resected, for example, only the saccule will respond to the caloric stimulation

In Table II, Group I can be considered as a control In Group II, the saccule is the only receptor It is notable that the change of nystagmus jerks in this group is reverse when it is compared with other groups Group III represents the effect of the utricular stimulation Group II and III indicate that a cold caloric stimulation on the utricle decreases the nystagmus while the same stimulation on the saccule increases the nystagmus jerks The last group shows that when all three receptors were destroyed, little or no change can be elicited by caloric stimulations

Considering the change of nystagmus pattern during the head motion, the effect of cold caloric stimulation to the utricle is represented by the change of the nystagmus during the increasing otolithic pressure on the utricular macula During a caloric stimulation of an ear in which the utricular macula is pressed the saccule will be placed in a similar condition

In our experiments (1960) it was found that the pressing stimulation on the saccular macula suppresses the nystagmus to the ipsilateral side, in other words increases the nystagmus to the contralateral side Group II in the second table varifies this statement

From those observations it can be concluded that the utricle and saccule respond to a caloric stimulation and acts as an inhibitory factor on the nystagmus of both direction, however, in the mode of action their effects on the nystagmus are antagonistic in nature

ZUSAMMENFASSUNG

Versuche an Kaninchen wiesen hin, dass die otolithischen Organe wahrscheinlich keinen Nystagmus durch die externale Stimulation produziren, aber sie gewissermassen regulatorisch die Entstehung des Nystagmus beeinflussen

Die Auf-und Abwartsbewegung des Kopfes und die kalorische Stimulation wurden auf die Tiere appliziert, die den durch die Resektion der Ampullarnerven hervorgerufenen Nystagmus hatten

Die nystagmographischen Registrierungen dieser Tiere, die horizontale Ampullarnerven, Utricularisnerven und Saccularisnerven wurden geschnitten, zeigten Anderungen in den Nystagmus bildern, die die Reaktion der otolithischen Stimulation aufwiesen Es wurde beobachtet, dass der Utriculus einen hemmenden Mechanismus auf den Nystagmus zur anderen Seite und der Sacculus einen auf den zur gleichen Seite leistete

REFERENCES

DIX, M R, and HALLPIKE, C S, 1952 The pathology, symptomatology and diagnosis of certain common disorders of the vestibular system *Proc Roy Soc Med*, *45*, 341

JONGKEES, L B W, 1950 Positional nystagmus of peripheral origin *J Physiol*, *110*, 447

———, 1950 On the function of the saccule *Acta oto laryng* *38*, 18

OWADA K, and SHIIZU, S, 1960 The eye movement as a saccular function *Acta oto-laryng*, *52* 63

OWADA, K, SHIIZU, S, and KIMURA, K, 1960 The influence of the utricle on nystagmus, *Acta oto laryng* *52*, 215

SULLIVAN, J A, JOHNSON, W, and SMITH, B, 1957 Normal and abnormal activity of the human otoliths *Ann Otol, etc, St Louis*, *66*, 918

ULRICH H, 1935 Die Funktion des Otolithen geprüft direkte mechanische Beeinflussung des Utriculusotolithen am lebenden Hecht *Pflug Arch ges Physiol*, *235*, 545

VERSTEEGH C 1927 Ergebnisse partieller Labyrinthexstirpation bei Kaninchen *Acta oto laryng* *11*, 393

35–Shinanomachi Shinjuku Tokyo

SOME OBSERVATIONS ON THE NEURONAL MECHANISM OF OPTO KINETIC NYSTAGMUS

NAOSHIGE MARUYAMA AKIO WATANABE
TADASHI KAWASAKI JUNICHIRO KOIZUMI
TAKASHI HIGUCHI and MASANORI MORIMOTO
Niigata Japan

From Brain Research Institute and Department of Otolaryngology (Head Prof M Morimoto M D) Niigata University School of Medicine Niigata

Unitary activities were recorded from the oculo motor nucleus and optic nerve in rabbits by means of super fine microelectrodes with the aim of clarifying the neuronal mechanism concerned in the reflex arc of optokinetic nystagmus

1) The unitary activities of oculo motor neurons when optic nystagmus was elicited were as follows

The discharge rate increased or decreased gradually upon rotating the optical cylinder until it reached a certain rate which continued to the beginning of nystagmus The impulse to the ocular muscle which rested in the quick phase decreased promptly to a minimum rate just before the beginning of the quick phase and increased gradually during the slow phase The change in the discharge rate of the impulses to the ocular muscle which contracted in the quick phase corresponded to the course of nystagmus

2) The eye speed in the slow phase of nystagmus which was recorded with electronystagmography was accelerated towards the nasal side when discharges of an optic nerve fiber which was presumed to be derived from the temporal side of the fovea were increased and it decelerated when discharges of an optic nerve fiber which was presumed to be derived from the nasal side of the fovea were increased

Thus the eye speed in the slow phase was controlled as the image of the stripe of the optical cylinder on the retina would be caught again by the fovea even if the fovea was turned away temporarily from the image

3) The responses of single optic nerve fibers to a shifting stripe were examined in curarized rabbits Some units reacted a different way according to the shifting direction of the stripe and some units reacted the same way In some units the responses were caused only by shifting in one direction

In our experiments, we intended to record unitary activities from various parts belonging to the reflex arc of opto-kinetic nystagmus with the aim of clarifying the neuronal mechanism concerned in that reflex

This paper is concerned with studies on the unitary activities recorded from the occulo motor nucleus and the optic nerve

Since most of the responses discussed in this study consist of very delicate changes in the unitary activities, the injury discharges or spontaneous changes of the activities often confused us in analyzing the data Therefore only when several, at least two, records of the same pattern were obtained from one and the same neuron, one of these records was brought into the discussion

METHOD

All experiments were performed on adult rabbits In order to elicit nystagmus the animals had to be awaked But under these conditions because of the animal's body movements it was impossible to keep a single neuron impaled with the microelectrode In order to avoid such disturbance the spinal cords of animals were severed at the level of L_3, and all their nerves to the forelegs were cut at the brachial plexus under Nembutal anaesthesia After the animal awaked the experiment was started The method described by Junk was used to elicit the opto kinetic nystagmus (Fig 1) the images of the stripes were projected to the inside of the cylinder-form screen and those stripes could be rotated



Fig 1 The apparatus for the experiment including elicitation of nystagmus

For recording unitary activities, glass capillary microelectrodes (about 0 2 μ of the tip diameter) filled with 3 M KCl were used The electrical resistances of these microelectrodes measured with 50 c/sec AC ranged from 15 to 25 MΩ The microelectrode was connected to the grid of a cathode-follower preamplifier The potential differences between the recording and the indifferent electrode were amplified with a condenser coupled (T C 0 03 sec) amplifier

Silver wire electrodes enclosed in a hypodermic needle were used for recording the electronystagmography These electrodes were inserted into the hypodermic tissue of the outer corners of both eyes The potential differences between both electrodes were amplified with a differential condencer coupled amplifier (T C 0 5 sec)

In order to examine the responses of those neurons to a moving stripe when the eyes were fixed, the animals were immobilized by the use of curare The apparatus for this experiment is shown in Fig 2

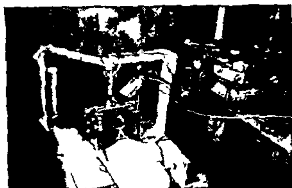


Fig 2 The apparatus for the experiment in the animals immobilized by the use of curare

A tube form bulb which was covered with black paper except for a slit was used as a stripe The slit was covered with several layers of filter paper to reduce the brightness which was made to be several lux. This bulb could be rotated and its position was indicated by the position of a beam on the face of the C R tube The rostral side is indicated as 0° and the caudal side as 180° in the following discussion

RESULTS AND DISCUSSION

1 *Unitary activities from the oculo motor nucleus when nystagmus was elicited*

The microelectrode was inserted into the oculo-motor nucleus through the upper colliculus When the eyes rested, the activity recorded from an oculo-motor neuron consisted of a succession of spikes with regular intervals The discharge rate, however, increased or decreased gradually upon rotating the stripes until it reached a certain rate which continued until the beginning of nystagmus

This phenomenon made us think that the eyes were kept in a deviated position in this stage The impulses to the ocular muscles which rested in the quick phase of nystagmus decreased promptly to a minimum discharge rate just before the beginning of the quick phase of the nystagmus and increased gradually during the slow phase On the other hand, the change in the rate of the impulses to the ocular muscles which contracted in the quick phase corresponded to the course of nystagmus

From these findings, it was revealed that before the muscles which would pull the eye in the quick phase was contracted at the beginning of the quick phase, the antagonists were relaxed

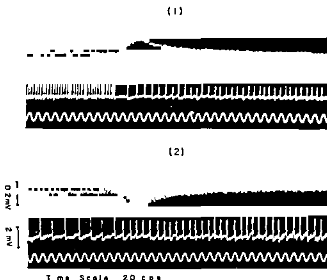


Fig 3 Unitary activities from the oculo motor nucleus during elicitation of nystagmus In each record Upper trace electronystagmography, middle trace unitary activity bottom trace time cal

2 *Unitary activities from the optic nerve when nystagmus was elicited*

For obtaining unitary activities from the optic nerve, the olfactory bulb was cut across in the middle and then the optic nerves were exposed by elevating the frontal part of the cerebrum The micro-electrodes were inserted into the peripheral part up to the chiasma

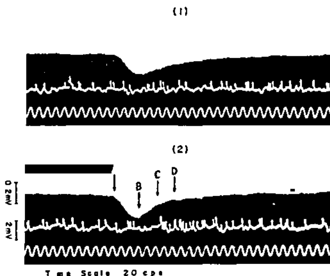


Fig 4 Unitary activity from the left optic nerve corresponding to optic nystagmus

Most of the unitary activities from the optic nerve did not show any change even when nystagmus was elicited Changes in the activities corresponding to the nystagmus were obtained from only a few units The records shown in Fig 4 are examples of such unitary activities recorded from the left optic nerve The rotating direction of the stripes was from the temporal side to the nasal side on the left These records were obtained successively from the same unit. In both records the angular velocity of rotation of the stripes was the same

Since the slow phase of the nystagmus in the upper record of Fig 4 is smooth, the eye might move in unison with the movement of a stripe

In Fig 5 the tracing of the slow phase of nystagmography in the upper record of Fig 4 which is shown by a broken line has been superimposed on the lower record of Fig 4 As the slow phase in the upper record of Fig 4 was considered to coinside with movement of a stripe, the broken line must be identifiable with the movement of the

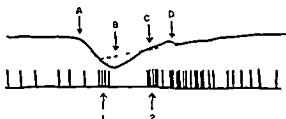


Fig 5 The tracing of the slow phase of nystagmography in the upper record of Fig 4, which is shown by a broken line has been superimposed on the lower record of Fig 4

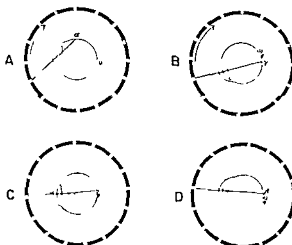


Fig 6 The positional relationship between the eye and the stripe at each time indicated as A B C and D in Fig 5

stripe It was supposed from Fig 5 that the movement of the eye showed a damped oscillation of which the center was the moving stripe

The unitary activity in the upper record of Fig 4 did not show any change even when nystagmus was elicited, but in the lower record high frequency discharges were observed when the eye got ahead of the stripe, such periods corresponded to the time when the tracing of nystagmography has been above the broken line in Fig 5

Fig 6, A B C and D show the positional relationship between the eye and the stripe at each time indicated as A B C and D in Fig 5

The image of the stripe on the retina had to be on the nasal side of the fovea when the eye got ahead of the stripe as indicated in Fig 6, C Since the discharges from this unit increased in such period, the receptive field from which this unit derives had to be on the nasal side of the fovea In the lower record of Fig 4 the eye speed in the slow phase was decelerated just after the beginning of high frequency discharges

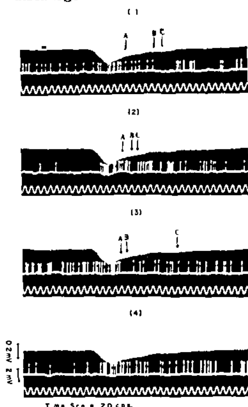


Fig 7 Unitary activity from the left optic nerve during elicitation of nystagmus The relationship between unitary activity and the course of nystagmography is opposite to that shown in Fig 4

In Fig 7, however, the relationship between unitary activity and the course of nystagmography was opposite to that shown in Fig 4

The discharge showed a marked increase when the eye was presumed to go behind the stripe and the eye speed in the slow phase was accelerated just after the beginning of high frequency discharges Therefore, the receptive field from which this unit derived had to be on the temporal side of the fovea

From the results mentioned above it was concluded that the increase in discharges of the unit derived from the nasal side of the fovea caused deceleration of the eye speed in the slow phase which turned towards the nasal side and an increase in discharge of the unit derived from the temporal side of the fovea caused acceleration of it When the eye got ahead of the stripe in the slow phase the eye

speed was decelerated, and the eye went behind the stripe the eye speed was accelerated Thus the eye speed in the slow phase was controlled as the image of the stripe on the retina would be caught again by the fovea

As mentioned above, the unit shown in Fig 4 was considered to be derived from nasal side of the fovea If so, the image of the stripe on the retina had to cross the receptive field in the quick phase But in the upper record of Fig 4 no change in discharge rate was observed during the quick phase In the lower record a burst of discharges was observed at the end of the quick phase but it was an exception This phenomenon made us think that the unit might react a different way according to the shifting direction of the stripe Perhaps the discharges of an optic nerve fiber derived from the region near the fovea may not show marked increase when the image crosses over the receptive field in its foveo petal shifting The increase in discharges of the units derived from the nasal side of the fovea was thought to cause the acceleration of eye movement towards the temporal side Therefore, if marked increase in the discharge of such units occurs at the end of the quick phase, the eye may run over the stripe because of the additional acceleration towards the temporal side Actually such a pattern was observed in the lower record of Fig 4 which was considered to be an exception Therefore, if the discharges of neurons derived from the region near the fovea are inhibited in foveo petal shifting of the image, such a mechanism may be convenient for the formation of the slow phase

3 *Unitary responses from the optic nerve to a shifting stripe when the eye was fixed*

In order to verify the fact that some units react a different way depending upon the shifting direction of the stripe, the responses of single optic nerve fibres to a shifting stripe (the slit on a tube form bulb c f Fig 2) were examined in curarized rabbits

Some units reacted a different way according to the shifting direction of the stripe and some units reacted the same way Fig 8 is an example of the latter

The response pattern did not depend upon the shifting direction of the stripe By shifting in both directions, initially it showed an inhibition of discharges and then an increase in discharges The stimulus of the moving stripe had almost the same effect on this unit when the light of the room was switched on and off rapidly This unit could not inform the centrum about the shifting direction of the object

Fig 9 shows another type of response

When the stripe was rotated from the nasal to the temporal side,

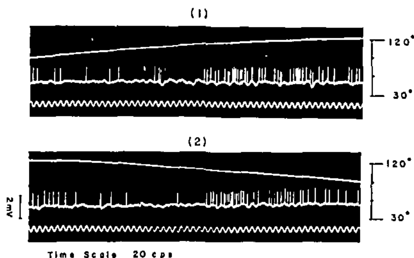


Fig 8 Unitary activity from the left optic nerve of the animal immobilized by the use of curare Both the records show the same response pattern although shifting direction of the stripe was opposite

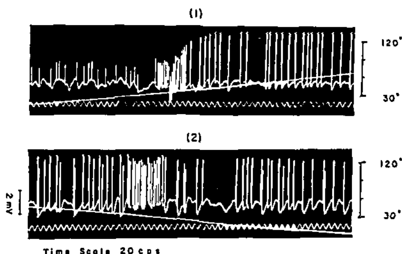


Fig 9 Unitary activity from the left optic nerve of the animal immobilized by the use of curare In the upper record the reaction consists initially of inhibition and then response, in the lower record initially discharges and then inhibition

initially an inhibition of discharges was observed and then an increase in discharges On the contrary, when it was rotated from the temporal to the nasal side, it showed the response and then the inhibition This unit might have the ability to inform the centrum of the object's shifting direction But the responses were caused by the shifting in both directions

In Fig 10 and Fig 11, however, the responses were caused only by shifting in one direction

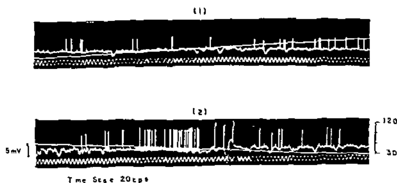


Fig 10 Unitary activity from the left optic nerve of the animal immobilized by the use of curare In the upper record no reaction is seen In the lower record a burst of discharges is evoked in response to the position of the stripe, 60°–50°

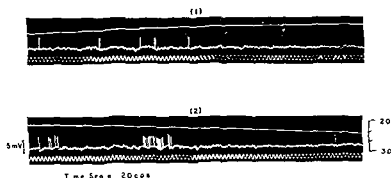


Fig 11 Unitary activity from the left optic nerve of the animal immobilized by the use of curare In the upper record no reaction is seen In the lower record response is seen when the stripe is at the position of 110°–105°

The left eye of the animal from which these records (Fig 10, Fig 11) were obtained was fixed as its papilla pointed in the direction of 66° In Fig 10 a burst of discharges was observed when the stripe crossed over the position of 60°–50° in the shifting towards the nasal side As the papilla pointed in the direction of 66°, the fovea was supposed to correspond to the position of between 60° and 65° Therefore, the receptive field of the unit from which the records of Fig 10 were obtained might be in the nasal side of the fovea From the positional relationship between the stripe and the fovea, it is clear that the shifting of the stripe towards the nasal side in the position of 60°–50°, in which the stimulus of a moving stripe caused the response, corresponded to the foveo-fugal shifting of the image on the retina This unit might be concerned in the formation of the slow phase

Fig 11 was obtained from another unit than that given in Fig 10 but from the same animal In Fig 11 the response was seen when

the stripe ran over the position of 110°–105° in the shifting towards the nasal side The position of 110°–105° was a peripheral part of the field of vision It is certain that the response was caused only by foveo petal shifting of the image This unit might not be concerned in the formation of the slow phase but in the formation of the quick phase Since the eye speed in the slow phase is controlled as the image of the stripe on the retina will be caught again by fovea even if the fovea is turned away temporarily from the image, the stripe never comes to the peripheral part of the field of vision It is widely accepted that the entrance of another stripe to the field of vision causes the quick phase of optokinetic nystagmus If it is true, the unit derived from the peripheral part of the retina must react to foveo petal shifting of the image of a stripe because entrance of a stripe to the field of vision always performs by foveo petal shifting of its image Since the unit of Fig 11 responded only to foveo petal shifting of the image and its receptive field was in the peripheral part of the retina, it was assumed to be concerned in the formation of the quick phase

It is concluded that the unit which shows such a response pattern as in Fig 10 may be concerned in the formation of the slow phase, and the unit as in Fig 11 may be concerned in the quick phase Perhaps the unit as in Fig 8 or Fig 9 may have no connection with the formation of nystagmus

ZUSAMMENFASSUNG

Die Aktivitat einzelner Neuronen wurden von dem okulo motorischen Kern und von N opticus in Kaninchen mit super-feinen Mikroelektroden registriert, um den im Reflexbogen des optokinetischen Nystagmus beteiligten neuronalen Mechanismus zu erklaren

1) Die Aktivitat einzelner Neuronen des Nucleus okulo motori, als optischer Nystagmus hervorgebracht wurde, war wie folgt

Die Entladungsfrequenz nahm allmahlich bei Umdrehung des optischen Zylinders zu oder ab, bis sie eine bestimmte Rate erreicht hat, die bis zum Beginn des Nystagmus dauerte

Die Impulse auf den Augenmuskel, in der raschen Phase bleibend, nahmen plotzlich eben vor dem Beginn der raschen Phase zu einer minimalen Rate ab und im Laufe der langsamen Phase nahmen sie al lmahlich zu Die Entladungsanderung des Impulses zum Augenmuskeln, der sich in der sich in der raschen Phase verkurzte, entsprach dem Verlauf des Nystagmus

2) Die Augengeschwindigkeit in der langsamen Phase des Nystagmus mit der Elecktronystagmographie registriert, wurde nach der nasalen Seite beschleunigt, als Entladungen einer Sehnervenfaser, die als abgeleitet von der temporalen Seite des Fovea vermutet wurde, zugenom

men hatten, und sie wurde deceleriert, als Entladungen einer Sehnervenfaser, die als abgeleitet von der nasalen Seite des Fovea vermutet wurde, zugenommen hatten Auf diese Weise wurde die Augengeschwindigkeit in der langsamen Phase kontrolliert, da das Bild des Streifens des optischen Zylinders auf der Netzhaut von der Fovea wieder gefangen wurde, auch wenn die Fovea einstweilig vom Bild abgewichen war

3) Die Aktivierung der Einzelfaser des N opticus auf dem kreisenden Streifen wurde in curarizierten Kaninchen untersucht Einige Neuronen regierten verschieden oder gleich der kreisenden Richtung des Streifens gemass In einigen Neuronen wurde die Aktivierung durch die Verschiebung nur in einer Richtung veranlasst

REFERENCES

ANDERSON S and GERNANDT, B E, 1954 Cortical projection of vestibular nerve in cat *Acta Oto laryng Suppl 116* 10

BÁRÁNY R, 1907 Weitere Untersuchungen uber den vom Vestibularapparat des Ohres reflektorischen ausgelosten rythmischen Nystagmus und seine Begleiterscheinungen *Mschr Ohrenh 41* 477

BÁRÁNY R, 1921 Zur Klinik und Theorie des Eisenbahner Nystagmus *Acta Oto laryng 3* 260

BARTELS M 1927 Ophthalmostatik und Ophthalmokinetik *Arch Ohrenh 118* 270

BRUNNER H, 1936 Über labyrinthare und okulomotorische Übererregbarkeit *Ver tenarzt 9* 244

ECKEL E 1954 Electrophysiologische und histologische Untersuchungen in Vestibulariskerngebiet bei Drehreizen *Arch Ohrenh 164* 487

ELDRED E and HAGBARTH K E, 1954 Facilitation and inhibition of gamma efferents by stimulation of certain skin areas *J Neurophysiol, 17* 59

GERNANDT B E and THULIN C A, 1952 Vestibular connection of the brain stem *Amer J Physiol 171* 121

GERNANDT B E and THULIN C A, 1953 Vestibular mechanismus of facilitation and inhibition of cord reflexes *Amer J Physiol, 171* 653

GERNANDT B E KATSUKI Y and LIVINGSTON R B, 1957 Functional organization of descending vestibular influences *J Neurophysiol 20* 453

HAGBARTH K. E, 1957 Excitatory and inhibitory skin areas for flexor and extensor motoneurons *Acta Physiol Scand. 26 Suppl* 94

HARTLINE H K 1938 The response of single optic nerve fibers of the vertebrate eye to illumination of the retina *Amer J Physiol 121* 400

KESTENBAUM A, 1930 Zur Klinik des optokinetischen Nystagmus *Arch Ophth 124* 339

KUFFLER S W, 1953 Discharge pattern and functional organization of mammalian retina *J Neurophysiol, 16* 37

LENNOX M A and MADSEN A 1955 Cortical and retinal responses to colored light flash in anesthetized cat. *J Neurophysiol 18* 412

VESTIBULAR INFLUENCE ON THE GAMMA EFFERENT MOTOR SYSTEM OBSERVED BY ANGULAR ACCELERATION

GENKICHI TOTSUKA*, MUTSURO SUZUKI*
and
KISOU KUBOTA**
Tokyo, Japan

**From the Department of Otorhinolaryngology (Head Prof T Ishikura), School of Medicine, Juntendo University Tokyo*
and
***Section of Neurophysiology, Institute of Brain Research, University of Tokyo, Tokyo*

Vestibular influence on the muscle spindle activity were studied in the decerebrated cats by recording single spindle discharges of the ankle flexors and extensors of both sides Both cupulometric and constant angular acceleration were applied as vestibular stimulation

1 Two different types of vestibular influence were observed in frequency of spindle discharge of the limb muscle One was non reciprocal facilitatory and the other was reciprocal These responses were the same as that observed by oscillatory rotation which was reported in the preceding paper

2 The reciprocal type response was predominant to non reciprocal facilitatory one, especially in decerebrated cat

3 Mechanism of vestibular control of muscle tone was discussed

INTRODUCTION

Since Flourens (1824) and Ewald (1892) described labyrinthine control on the muscle, many researchers had been accumulated concerning reflex natures of labyrinth origin, forced postures after labyrinthotomy or in inner ear diseases and concerning clinical methods to examine labyrinthine muscle tone Recently, it was shown that the gamma efferent motor system described at first by Leksell (1945), is influenced from supraspinal structures and its role in motor control through proprioceptive loop was emphasized by Eldret et al (1953) Therefore, vestibular motor control has to be re-examined in view of the gamma motor system, and to be distinguished direct influence to motor neurons from indirect one which is modulated by motor activity and muscle spindle activity The attempt along this line was initiated by Andersson and Gernandt (1956) who showed that small gamma efferents were activated at a lower strength of electrical labyrinthine stimulation than the alpha fibers The authors reported in the preceding paper that activity of the gamma system was

influenced by vestibular stimulation The purpose of the present paper is to show the influence evoked by rotatory stimulation of one direction

METHODS

Under ether anesthesia cat was decerebrated vertically at the levels of Fr 2 0—10 0 (Jasper's *Atlas) after bilateral ligation of the common* carotid artery Animals were mounted in prone position on stereotaxic instrument of Todai-Noken type designed by Tokizane After lumbar laminectomy dorsal roots were cut bilaterally at the entry from L_6 to S to avoid reflex influences Both hind limbs were fixed to the instrument at upper and lower ends of the femur and at the ankle joint. Single spindle discharges of the ankle flexors and extensors (M tibialis anterior and M extensor digitorum longus, M gastrocnemius lateralis et medialis, M soleus and M plantaris) were isolated from dorsal root filaments *Single discharges were identified as muscle spindle activity of a given* muscle by the resting pause of the discharge after motor nerve stimulation and by muscle stretch (G Ia and G II fibers were not differentiated) Muscles were freed from bones at distal tendons and separated each other as possible Nerves of hind limbs except above mentioned muscles were cut Changes of spindle activity in frequency was attributed to change of the gamma efferent activity only in the absence of muscle contraction Muscles were maintained under steady stretch of constant strength throughout the experiment Spindle discharges were projected on four-beam cathode ray oscilloscope and recorded by kymograph camera Muscle contraction were observed by four channel electroencephalograph electromyographically recording with two steel needles inserted in a testing muscle

As natural stimulation of the labyrinth, two kinds of angular acceleratory stimulation on horizontal plane were used The first was the cupulometric stimulation After subliminal acceleration animals were subjected to constant rotation of 15°—120°/sec for two minutes until steady spindle activity was attained and rotation was stopped The rotation was recorded photoelectrically The second kind of stimulation was the rotation with constant angular acceleration of $1°—8°/sec^2$ The axis of rotation in each experiment was carefully set at the middle point between both ears Precautions were paid to exclude any stimulus except the given rotation which may influence spindle activity or any mechanical and electrical disturbance

RESULTS

1 *Cupulometric stimulation*

Muscle spindle activities of both flexor and extensor were changed by the sudden stop of constant rotation, both clockwise and counter-clock-

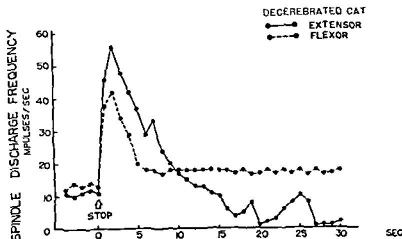
wise As illustrated in diagrams of Fig 1, spindle discharges of l flexor (●---- ●) and l extensor (●——●) increased their frequency by clockwise (upper diagram) and counter clockwise rotation (lower) after the moment of stopping The augmentation reached at its peak within a few second after stop and decreased gradually towards the control frequency in case of clockwise acceleration of flexor spindle But in extensor clockwise combination and flexor counterclockwise combination decrease lower than the initial level was observed Diminution of frequency without any augmentation was not observed Increase and appearance of spindle spikes after cupulometric stimulation was shown in Fig 2, together with the mechanical record of the rotation Relation between the response of spindle activity and acceleratory stimulus intensity which was represented by angular velocity final was illustrated in Fig 3 Intensity of the stimulus raised, peak frequency of the spindle augmentation increased

2 *Constant angular accelerated stimulation*

Spindle activity of l flexor and r extensor was augmented during counter-clockwise acceleration, and that of l extensor and r flexor was augmented during clockwise This augmentation was consistent, but reciprocal inhibitory change of antagonistic muscles was occasionally observed and the decrease was often accompanied by a initial temporary increase Relation between stimulus and the increase of spike frequency was shown in Fig 4 from the spindle of l extensor during counter-clockwise acceleration of 1°, 2°, 4° and 8°/sec^2 On the contrary in flexor spindles linear relationship was not observed during counter-clockwise acceleration

DISCUSSION

Two different types of vestibular influence upon the muscle spindle activity are already demonstrated in the author's preceding report, in which sinusoidal oscillatory rotation on the horizontal plane is applied on animals as natural stimulation of the labyrinth One of the types is simultaneous and non-reciprocal influence showing augmentation in flexor and extensor of both sides The other is reciprocal showing periodic and antagonistic changes between flexor and extensor corresponding to the acceleration While non-reciprocal pattern is observed as increased level of discharge frequency from the beginning of the ocillatory rotation to its end, the reciprocal pattern as periodical shift of frequency The failure to show reciprocal pattern as a clear change in cupulometric and constant angular acceleration as mentioned in " results " in contrary to oscillatory rotation may suggest that reciprocal pattern were masked by simultaneous non-reciprocal augmentation because of the latter's strong influence from the labyrinth It may be considered, moreover, that the non reciprocal pattern is predominant against reciprocal in decerebrated state



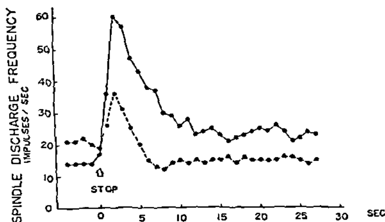


Fig 1 Interval diagram showing discharge interval of single spindle efferent from left flexor (●----●) and extensor (●——●) during cupulometric stimulation The rotation is clockwise and 120°/sec. in upper figure and counterclockwise in lower figure Spindle discharge of both muscles is increased abruptly after stop of the rotation towards both direction. While gradual decrease of discharge frequency until its initial level is observed after a few second augmentation in extensor-clockwise and flexor-counter-clockwise combination, rapid decrease until lower level than the initial is in flexor-clockwise and extensor-counterclockwise combination

Ordinate of figures represents spike numbers per second measured at consecutive every one second. Abscissa time after stop in second.

wise As illustrated in diagrams of Fig 1, spindle discharges of l flexor (●---- ●) and l extensor (●——●) increased their frequency by clockwise (upper diagram) and counter clockwise rotation (lower) after the moment of stopping The augmentation reached at its peak within a few second after stop and decreased gradually towards the control frequency in case of clockwise acceleration of flexor spindle But in extensor-clockwise combination and flexor-counterclockwise combination decrease lower than the initial level was observed Diminution of frequency without any augmentation was not observed Increase and appearance of spindle spikes after cupulometric stimulation was shown in Fig 2, together with the mechanical record of the rotation Relation between the response of spindle activity and acceleratory stimulus intensity which was represented by angular velocity final was illustrated in Fig 3 Intensity of the stimulus raised, peak frequency of the spindle augmentation increased

2 *Constant angular accelerated stimulation*

Spindle activity of l flexor and r extensor was augmented during counter-clockwise acceleration, and that of l extensor and r flexor was augmented during clockwise This augmentation was consistent, but reciprocal inhibitory change of antagonistic muscles was occasionally observed and the decrease was often accompanied bv a initial temporary increase Relation between stimulus and the increase of spike frequency was shown in Fig 4 from the spindle of l extensor during counter-clockwise acceleration of 1°, 2°, 4° and 8°/sec ^{2} On the contrary in flexor spindles linear relationship was not observed during counter clockwise acceleration

DISCUSSION

Two different types of vestibular influence upon the muscle spindle activity are already demonstrated in the author's preceding report, in which sinusoidal oscillatory rotation on the horizontal plane is applied on animals as natural stimulation of the labyrinth One of the types is simultaneous and non reciprocal influence showing augmentation in flexor and extensor of both sides The other is reciprocal showing periodic and antagonistic changes between flexor and extensor corresponding to the acceleration While non-reciprocal pattern is observed as increased level of discharge frequency from the beginning of the ocillatory rotation to its end, the reciprocal pattern as periodical shift of frequency The failure to show reciprocal pattern as a clear change in cupulometric and constant angular acceleration as mentioned in " results " in contrary to oscillatory rotation may suggest that reciprocal pattern were masked by simultaneous non reciprocal augmentation because of the latter's strong influence from the labyrinth It may be considered, moreover, that the non-reciprocal pattern is predominant against reciprocal in decerebrated state

In the studies on deviation of the arm and its electromyographic analysis, labyrinthine tonus change of the arm muscle in non reciprocal type has not been considered to be significant, however, non reciprocal augmentation to the gamma system observed in present study suggests its function such as to produce a preparatory background state to alpha motor neurons through gamma loop, giving the bias to spindle activity Therefore, direct vestibular influence to alpha motor system may operate securely in integrated manner together with reciprocal influence to gamma system

ACKNOWLEDGEMENTS

The authors wish to express their gratitudes to Professor Toshihiko Tokizane of Brain Research Institute, Tokyo University and Professor Takeo Ishikura, Department of Medicine, Juntendo University and also to members of Tokizane School for their advices during the experiments and in the preparation of this paper

ZUSAMMENFASSUNG

Vestibulare Einflusse auf die Aktivitat des Muskelspindels werden in den enthirnenden Katzen durch die Registrierung des aus dem einzigen Spindelfaser des beiderseitigen extensierenden und flexierenden Fussgelenkmuskels gefuhrten Aktionsstroms Als vestibulare Reizung wurden beide der cupulometrischen bzw konstant beschleunigenden Methode angewandt

1 Die Haufigkeit des Aktionsstroms aus der Spindelfaser des Unterschenkelmuskels wurde vestibular in zwei Typen beeinflusst Der eine war der nicht reciprokale fascilitatorische und der andere war der reciprokale Diese Reaktionstypen waren derjenige als die Beobachtung durch die oscillatorische Rotation, die im letzten Blatt berichtet wurden

2 Der reciprokale Reaktionstyp war merkwurdiger als der nichtreciprokale fascilitatorische, besonders in der enthirnenden Katze

3 Der Mechanismus der vestibularen Regulierung des Muskels wurde erortert

REFERENCES

ANDERSSON S and GERNANDT B E, 1956 Ventral root discharge in response to vestibular and proprioceptive stimulation *Neurophysiol 19* 524

ELDRED E GRANIT R and MERTON P A, 1953 Supraspinal control of the muscle spindles and its significance *J Physiol 122* 498

EWALD J R, 1892 *Physiologische Untersuchungen uber das Endorgan des Nervus Octavus* Wiesbaden, Bergmann.

STOP 1 SEC

Fig 2 Spindle discharge during cupulometric stimulation by clockwise rotation The upper line indicates spike discharge of single spindle efferent of left flexor, the middle line that of flexor, and the lower line indicates rotation of the turning appaiatus recorded by photoelectrical method

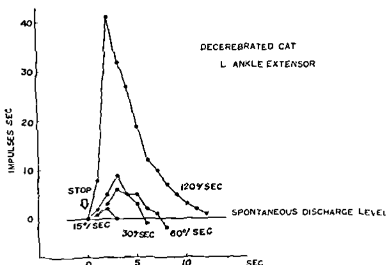


Fig 3 *Interval diagram of the spindle discharge of left flexor after cupulometric stimulation of clockwise rotation* Its final velocity is 120°, 60°, 30° and 15° /sec Parallel relation between the response of spindle activity and acceleratory intensity is shown

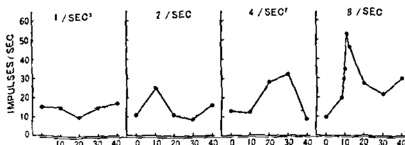


Fig 4 *Interval diagrams of the spindle discharge of left flexor* during constant angular acceleration of clockwise direction The a-celeration is 1°, 2°, 4° and 8°/sec² Parallel relation between responses of spindle efferent and the intensity of acceleration is shown in acceleration over than 2°/sec²

In the studies on deviation of the arm and its electromyographic analysis, labyrinthine tonus change of the arm muscle in non reciprocal type has not been considered to be significant, however, non reciprocal augmentation to the gamma system observed in present study suggests its function such as to produce a preparatory background state to alpha motor neurons through gamma loop, giving the bias to spindle activity Therefore, direct vestibular influence to alpha motor system may operate securely in integrated manner together with reciprocal influence to gamma system

ACKNOWLEDGEMENTS

The authors wish to express their gratitudes to Professor Toshihiko Tokizane of Brain Research Institute, Tokyo University and Professor Takeo Ishikura, Department of Medicine, Juntendo University and also to members of Tokizane School for their advices during the experiments and in the preparation of this paper

ZUSAMMENFASSUNG

Vestibulare Einflusse auf die Aktivität des Muskelspindels werden in den enthirnenden Katzen durch die Registrierung des aus dem einzigen Spindelfaser des beiderseitigen extensierenden und flexierenden Fussgelenkmuskels gefuhrten Aktionsstroms Als vestibulare Reizung wurden beide der cupulometrischen bzw konstant beschleunigenden Methode angewandt

1 Die Häufigkeit des Aktionsstroms aus der Spindelfaser des Unterschenkelmuskels wurde vestibulär in zwei Typen beeinflusst Der eine war der nicht reciprokale fascilitatorische und der andere war der reciprokale Diese Reaktionstypen waren derjenige als die Beobachtung durch die oscillatorische Rotation, die im letzten Blatt berichtet wurden

2 Der reciprokale Reaktionstyp war merkwurdiger als der nichtreciprokale fascilitatorische besonders in der enthirnenden Katze

3 Der Mechanismus der vestibularen Regulierung des Muskels wurde erortert

REFERENCES

ANDERSSON S and GERNANDT B E 1956 Ventral root discharge in response to vestibular and proprioceptive stimulation *Neurophysiol 19*, 524

ELDRED E, GRANIT R and MERTON P A, 1953 Supraspinal control of the muscle spindles and its significance *J Physiol 122* 498

EWALD J R, 1892 *Physiologische Untersuchungen uber das Endorgan des Nervus Octavus* Wiesbaden, Bergmann

EWALD, J R, 1894 Wirkung des Labyrinthtonus der Zugkurve des Muskels *Deutch Med Wochenschr*, *20* Tg 69
FLOURENS, M, 1824 *Recherches experimentales sur les proprietes du sumpteme nerveux animaux vertebres* Paris
GERNANDT, B, 1960 Vestibular Mechanisms, *Handbook of physiology*, *Vol 1 of Section I*, 549
KUBOTA, K, SUZUKI, M and TOTSUKA, G Labyrinthine Influence on the muscle Spindle activity *Jap J Physiol* in preparing
LEKSELL, L, 1945 The action potential and equitatory effects of the small ventral root fibers on skeletal muscle *Acta Physiol (Scand) 10, Suppl 31* 1
SHIMAZU, H, HONGO, T and KUBOTA, K, 1962 Two types of central influence on gamma motor system *J Neurophysiol 25*, 309

Yushima Bunkyoku, Tokyo

HEAD MOVEMENTS OF THE VESTIBULAR ORIGIN AND THE REGULATORY FUNCTIONS OF THE CENTRAL NERVOUS SYSTEM

SADAO UTSUMI, HIROSHI SHINDO, TEIZO MUKAI
and YASUTERU YAMANAKA
Nara, Japan

From the Department of Otorhinolaryngology (Head Prof S Utsumi, M D) Nara Medical College, Nara

Changes in head movements and the behaviours of various neck muscles which were caused by unilateral vestibular stimuli were studied electronystagmoraphycally in rabbits The results obtained are as follows 1) When the head is deviated on the side of a non stimulated ear, there immediately develops an increase in the tone of the perivertebral neck muscles of the same side In head nystagmus the bilateral superficial anterior neck muscles which operated alternately head movements are inhibited on each side play the most important role 2) The vestibular head movements are inhibited in consequence of a marked increase of tonus in the superficial nape muscles of the stimulated side under the control of the cerebellum

INTRODUCTION

Flourens (1824) reported characteristic postural changes resulting from unilateral labyrinthectomy in pigeons Ewald, Magnus, Hasegawa and many other workers have studied vestibular head movements from morphological, physiolosical and histological points of view

Since 1952, in our laboratory vestibular muscle tonus at rest and muscle responses induced by various kinds of stimulation on the vestibulum have been studied electromyographically Histological studies have been also made on the muscles in the ocular, cervical, dorsal and extremity *regions in rabbits*

In the present experiment the authors studied muscle activities and the regulatory function of higher nervous centres especially of the cerebel lum which controls the vestibular head movements in various neck muscles

METHOD

Healthy mature rabbits were fixed on a board and were decerebrated and decerebellated

The animals' head movements develop following unilateral vestibular stimulation In order to cause head deviation galvanic stimuli (4 2 V,

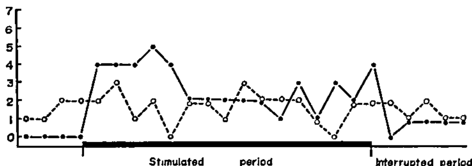


Fig. 1. Frequency diagrams of discharges on the basis of the EMG in deep nape muscles following a cathodic stimulation in normal rabbit, beeing plotted the numbers of discharges in each 1/6 second. ●——● In the muscle of non-stimulated ear side O--O In the muscle of stimulated ear side. (The same shall be applied to the following)



Fig. 2 Frequency diagrams of discharges in deep anterior neck muscles following a cathodic stimulation in normal rabbit.

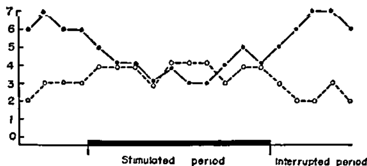


Fig 3 Frequency diagrams of discharges in deep anterior neck muscles through an anodal stimulation in normal rabbit.

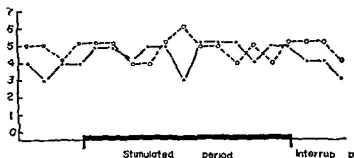


Fig 4 Frequency diagrams of discharges in superficial nape muscles at the terminal stadium owing to repeated stimuli in normal rabbit.

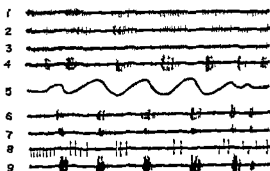


Fig 5 In normal rabbit.

Fig 5, 6 Schematic illustrations of electromyographic changes in neck muscles when head nystagmus is occured by a slight positive pressure *on the fenestrated unilateral* horizontal canal 1–4 Non stimulated ear side 5 Mechanogram of head movement 6–9 Stimulated ear side 1 6 = M rect capit. 2,7 = M long 3,8 = M trapz 4,9 = M sternomast.

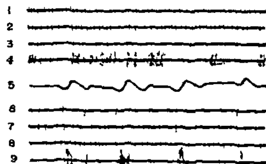


Fig 6 After the removal of the contralateral cerebral hemisphere and the cortical part of the ipsilateral cerebellar hemisphere on the basis of a stimulated ear

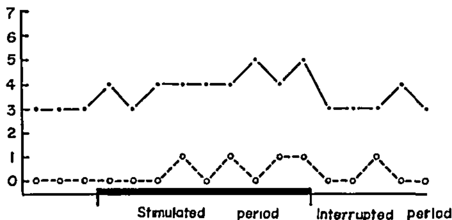


Fig 7 Frequency diagrams of discharges in superficial nape muscles in the animal which has been decerebrated and removed the vermis cerebelli, at the terminal stadium owing to a repeated cathodic stimuli

DISCUSSION

The stimuli given to the vestibulum are classified into two kinds, *one is so-called physiological and the other is non-physiological* The former is rotatory or linear progressive movements on the horizontal and vertical axes given to both vestibula, and the latter is galvanization, pressure and calorization given to an unilateral vestibulum Since Hitzig (1871) the head movements due to galvanic stimulation through the vestibulum have been studied by Breuer, Max, Ishihara, Oda, Dohlmann, Wittmaack and others The head movements induced by pressure on the fistular lesion in the ear of a patient were reported by Urbantschitsch (1910) The finding, being designated 'fistulous symptom", has been studied by Scheibe, Ruttin, Hofer, Baldenveck and Block, Mygind, Kragh, etc

Recently electromyography was used in measuring muscle tonus and responses of labyrinthine orgin by Tokizane, van Eyck, Takagi, Morimoto, Fukuda, Honjo, etc Van Eyck (1953) made a study on the labyrinthine muscle reflex of the neck in the pigeon on an electromyographical basis

We reported in our previous papers that the muscle tonus in the deep nape is governed by labyrinthine tonus contralaterally at rest and that the head deviated to the opposite side when subjected to positive stimuli on a unilateral vestibulum in order to increase the muscle activity of the deep nape on the deviated side

In the present experiment non-physiological stimuli, i e, very weak galvanization and pressure were given to a unilateral vestibulum The responsive activity of the neck in relation to the vestibular head deviation

and nystagmus was confirmed sufficiently in the perivertebral (the deep nape and deep anterior neck), superficial nape and superficial anterior neck muscles The head deviation was the fundamental reflex of a vestibular origin, as a result from the changes in the tonic activity of the perivertebral muscles of the contralateral side M sternomastoideus showed the most significant activity for the head nystagmus

During repeated stimulation for head deviation and the development of head nystagmus, the regulatory function under the control of the higher nervous centres was observed Thus the rigidity of the superficial nape muscles, especially on the side of the stimulated ear, increased in order to inhibit head movements The inhibitory function against head deviation was mainly evoked by the vermis cerebelli and that against head nystagmus was controlled mainly by the cerebellar hemisphere

ZUSAMMENFASSUNG

Die Tatigkeitszustande der Kopfbewegungen und der Halsmuskeln, die durch den Reiz im unilateralen Vestibulum eines Kaninchens verursacht werden, haben wir mittels des Electromyograms erforscht Die Errungenschaften der Forschung sind wie folgt

1) Im Falle der Deviation eines Kopfes zur contralateralen Seite des Ohres wird der Tonus der perivertebralen Halsmuskeln rasch verstarkt, dagegen im Falle des Kopfnystagmus sind die superficiellen Vorderhalsmuskeln auf beiden Seiten wechselweise in Wirksamkeit, welche die Hauptrolle spielen

2) Diese vestibularen Kopfbewegungen werden durch die starke Tonuszunahme der superficiellen Nackenmuskeln auf der ipsilateralen Seite des Ohres gehemmt und diese Hemmungsfunktion steht immer unter der Herrschaft des Kleinhirns

REFERENCES

VAN EYCK M 1953 Étude eletromyographique des reflexes labirinthiques cerviceaux du pigeon *Acta oto laryng 43* 303

FLOURENS M J P 1824 *Recherches experimentales sur les proprietes et les fonctions du systeme nerveux dans les animaux vertebres* Crevot, Paris

HITZIG E 1871 Über die beim Galvanisieren des Kopfes entstehenden Storungen der Muskelinnervation und der Vorstellungen von Verhalten im Raume. *Archiv fur Anatomie Physiologie und wissenschaftliche Medicin Jahrg 1871*, 716

MOUKAI T 1958 Études des relations entre le labyrinthe et lobule petrosus cerebelleux chez le lapin *Med. J of Osaka Univ 8* 571

URBANTSCHISCH E 1910 Kopfnystagmus *Mschr f Ohrheilk 44* 1

UTSUMI S SHINDO H ISHIKAWA S MOUKAI T et YAMANAKA, Y 1957 Étude electromyographique du reflexe tonique labyrinthique *Med J of Osaka Univ 7*, 971

ON THE SECONDARY PHASE OF NYSTAGMUS

MASANORI MORIMOTO KANEMASA MIZUKOSHI, TSUTOMU OTANI, SHIGERU IKEDA, YU KATSUMI, TSUNEO SASAKI and YOSHIO KOIKE
Niigata Japan

From the Department of Otolaryngology (Head Prof M Morimoto, M D) Niigata University, School of Medicine, Niigata

Under various conditions, the authors have sought the secondary phase of vestibular and optokinetic nystagmus evoked in normal human subjects and rabbits Results obtained are as follows

1) The secondary phase of nystagmus can be elicited easily even with rather weak *stimuli as used clinically regardless of whether caloric or* optokinetic stimuli were employed If it cannot be observed, it is because of extinction due to inhibitory factors such as visual inputs or unknown effects

2) In the authors' opinion, the secondary phase of nystagmus is organized by primitive tracts in the brain stem and is constantly influenced and controlled by the higher vestibular and optokinetic nystagmus tracts such as the cerebrum or cerebellum

3) In rabbits, the authors have experienced some cases in which optokinetic nystagmus is transformed into eye movements resembling so called 'Nystagmus Clonus' or 'Firing" which become completely independent of the original stimuli in regard to the frequency of beats The presence of this phenomenon supplies us counter evidence opposing Barany's theory concerning the genesis of optokinetic nystagmus

Since Barany referred to the secondary phase of postrotatory nystagmus and gave a name "Nach-Nach-Nystagmus" in a paper published in 1907, efforts have been made by several authors to analyze this phenomenon All the investigations previously reported, however, have been limited in observations of the secondary phase of postrotatory nystagmus, without persuing in parallel observations of that of caloric or optokinetic nystagmus And yet, up to the present time almost no report is available on the investigation concerning the relationship between the secondary phase of vestibular and optokinetic nystagmus Therefore, the theories put forward in an attempt to explain the mechanism responsible for the occurrence of this phenomenon are at a considerable variance and still hypothetical For this reason, the present investigations have been undertaken to confirm and systematize our existing information upon the obscure points involved and to obtain further experimental data concerning its nature and physiological mechanism Under various conditions we have sought the secondary phase of vestibular and optokinetic nystagmus evoked in normal human subjects and rabbits

EXPERIMENTAL METHODS AND RESULTS

The recording of nystagmus was performed in all test by means of electronystagmography which will be referred to as ENG in this paper Rotatory stimuli were administrated by the rotary cabinet with an electrical driven unit The rotary stimulation consisted of accelerations of 0 1°—2 4°/sec² or of constant speeds of 60°—180°/sec for periods of 1—2 min In the caloric tests the subjects were seated with their head anteverted 30°, while the animals were held on a board with their head inclined upward to bring the external canal into the optimal position for stimulation Calorization was performed with 20—100 cc of water of 20°—30°C in the human subjects while in rabbits with 20 cc of water of 5°—10°C with the eyes closed In the optokinetic tests the subjects were seated inside a cylinder controlled by an electrically driven motor This cylinder which is 1 m in height and 1 5 m in diameter consists of 8—16 vertical stripes alternating with white spaces The speed of the revolving drum was kept with accelerations of 0 1°—1 0°/sec² for a period of 2 min, and suddenly brought to rest at a certain velocity Simultaneously, all visual inputs were eliminated, but ENG was uninterruptedly recorded Figs 1, 2 and 3 demonstrate typical recordings of the whole period of primary and secondary phases in vestibular and optokinetic nystagmus evoked in normal human subjects

As shown in Figs 1, 2 and 3, the secondary phase was clearly recorded always on the opposite side of the primary phase In all subjects tested, however, the secondary phase was not always recorded It was sometimes impossible to record the secondary phase, especially in caloric nystagmus The incidence of the secondary phase varies greatly according to individual

Secondary Phase of Postrotatory Nystagmus
(Human Subject)

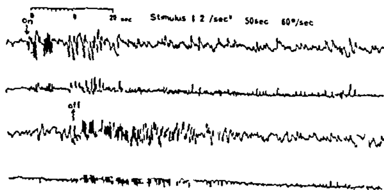


Fig 1

Secondary Phase of Caloric Nystagmus
(Human Subject)

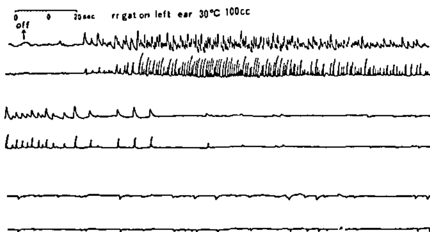


Fig 2

Secondary Phase of Optokinetic Nystagmus
(Human Subject)

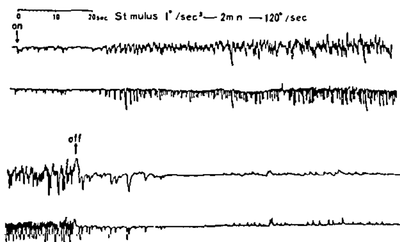


Fig 3

and species difference or to difference in the character of nystagmus as shown in Table 1

Tables 2, 3 and 4 show the latency of the secondary phase after the cessation of the primary phase and the duration and number of secondary nystagmus in human subjects measured from ENG recording

The presence of illumination and visual inputs affected significantly the occurrence of the secondary phase Usually, the secondary phase was best manifested and enduring with the lights off, but its duration became shorter or completely disappeared with the lights on Turning on the lights and visual inputs was followed by its prompt disappearence The

Table 1

THE RATE OF OCCURRENCE OF SECONDARY NYSTAGMUS

	Human Subject	Rabbit
Rotatory Nystagmus	100	20%
Caloric Nystagmus	60	60–70%
Optokinetic Nystagmus	100	100%

Table 2

ROTATORY NYSTAGMUS

Subjects	Stimulus		Postrotatory Nystagmus		Interval	Secondary Phase	
			Duration	No. of Beats		Duration	No. of Beats
1	180° sec² → 30	R	46	42	11	110	34
		L	43	38	16	92	28
2	0.4° sec² → 28 sec	R	52	39	61	19	7
		L	54	43	57	24	5
3	1.2° sec² → 60 sec	R	33	45	22	108	30
		L	36	55	36	74	38
4	2.4° sec → 36 sec	R	41	36	48	63	13
		L	44	41	51	52	10

Table 3

CALORIC NYSTAGMUS

Subjects	Stimulus		Primary Phase		Interval	Secondary Phase	
			Duration	No. of Beats		Duration	No. of Beats
1	25°C 100 cc	R	221	170	46	353	43
		L	202	153	40	361	54
2	15°C 00 cc	R	258	362	52	181	82
		L	273	386	68	170	76
3	25°C 20 cc	R	132	201	42	49	21
		L	148	218	35	56	28
4	30°C 100 cc	R	168	209	13	186	28
		L	182	224	9	210	33

Table 4

OPTOKINETIC NYSTAGMUS

(St mulus 1°/sec²—2m n — 120 /sec)

Subjects		After Nystagmus		Interval	Secondary Phase	
		Durat on	No of Beats		Durat on	No of Beats
1	R	327	190	23	461	65
	L	453	310	94	147	33
2	R	43	29	61	134	73
	L	63	42	47	126	44
3	R	30	29	8	18	10
	L	6	6	3	19	10
4	R	284	170	39	198	36
	L	189	113	24	247	36

amplitude and eye speed of the secondary phase evoked in human subjects were relatively small and low Therefore sometimes it was impossible to recognize the secondary phase Repeated optokinetic or caloric stimulation promoted the occurrence of the secondary phase and shortened the intervals between the primary and secondary phases In rabbits, when accelerative optokinetic stimuli were repeated with short intervals, there were some cases in which evoked nystagmus were transformed into eye movements resembling so called " Nystagmus Clonus " or " Firing " which became completely independent of the original stimuli in regard to the frequency of beats as shown in Fig 4

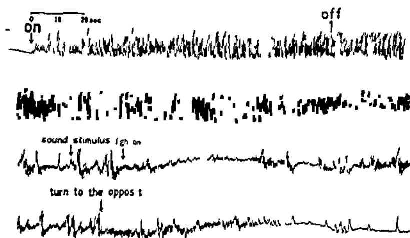


Fig 4

Even when the drum was stopped, this "Firing" phenomenon lasted for a long period of time until it finally disappeared by a reverse optokinetic stimulation This phenomenon was more easily elicited after extirpation of the bilateral cerebral hemispheres If the drum was stopped just before the onset of "Firing", the secondary phase was more easily elicited and lasted for a long period of time We have studied influences of the central nervous system upon the occurrence of the secondary phase in rabbits in which various operative procedures have been done on their central nervous system The results of the experiment are summarized below

Firstly, destruction or extirpation of the bilateral cerebral hemispheres promotes the occurrence of the secondary phase, especially in postrotatory nystagmus Secondary, destruction or extirpation of the unilateral cerebral or cerebellar hemisphere and unilateral destruction of paraflocculus in the cerebellum elicites a directional preponderance (D P) to the operated side, and consequently increase the occurrence of the secondary phase to D P side, while those to sound side decrease or disappear It is of interest that a correlation does exist between the threshold of the occurrence of the secondary phase of postrotatory and caloric nystagmus, i e, the lower the threshold of postrotatory secondary nystagmus, the lower the threshold of the caloric one as shown in Fig 5

The authors have firstly presumed the existence of this correlation also in optokinetic nystagmus, but no decisive conclusion could be obtained

CORRELATION BETWEEN THRESHOLDS OF SECONDARY PHASE OF ROTATORY AND CALORIC NYSTAGMUS



Fig 5 The number in ordinate shows the minimal angular speed necessary for the occurrence of secondary phase of postrotatory nystagmus The number in abscissa shows the minimal temperature (difference between the temperature of douching and the subject's temperature) necessary for the occurrence of secondary phase of caloric nystagmus.

because of a number of complicated factors For all that it is reasonable to consider that the higher the sensitivities of the vestibular and optokinetic systems, the greater the occurrence of secondary phase This consideration will be acceptable in view of the fact that in some patients with diseases of the central nervous system showing abnormal hypersensitivity to optikinetic and vestibular stimuli the secondary phase was more clearly elicited and enduring than the forgoing primary phase

COMMENTS

Of all our experimental facts, one significant observation is that the secondary phase can be elicited easily even with rather weak stimuli such as clinical tests regardless of whether caloric or optokinetic stimuli were employed Up to the present day it is said that the secondary phase of caloric nystagmus is only observed by particular calorization such as Hot/Cold double irrigation (Aschan 1955), continuous douching lasting for 20—40 min (Wagemann 1958) or two-staged caloric test (Boninghaus 1953) The authors, however, have obtained different data This is possibly because of the difference in method of observation, i e, naked eyes and ENG recording As for the origin of the secondary phase of nystagmus, there exist at present two view quite opposite to each other The one attempts to ascribe the mechanism of the secondary phase to an interplay of some central neural agency, while the other seeks it in the cupular organ The former was supported by Fischer and Wodak 1922, Fischer 1928, Veits 1931, Lange 1939, Schmalz 1932, Mittermaier 1938, Aschan 1955 and Mittermaier and Rossberg 1956, the latter by van Egmond, Groen and Jongkees 1952 In connection with reports of caloric secondary nystagmus, little information is available Firstly, Fischer and Wodak reported on rhythmic sensations of vertigo following caloric stimulation Then, Thornval 1936, Jung 1953, Boninghaus 1953, Aschan 1955, Stahle 1958, Hamersma 1955, McLay, Madigan and Ormerod 1958 also reported on them Even these authors observed caloric secondary nystagmus only by a chance or by special methods of calorization as mentioned before Even in central genesis theory, there are a number of different view For example, Aschan (1955) from his experimental results of Hot/Cold double irrigation, assumed that the secondary phase was due to a central summation Jung (1953) attempted to ascribe it to " Postexcitatorische Hemmungsphase " On the contrary, van Egmond, Groen and Jongkees considered that the genesis of secondary nystagmus could equally be accounted for by peripheral processes, not by oscillation in the cupula, but by electrical phenomenon or elastic after effects in the nondeflected cupula As for the reports of optokinetic secondary nystagmus, there were found in the literature only those of Gruttner 1939, McLay, Madigan and Ormerod 1958 and Mackensen 1958 However, in these reports, they

observed principally the presence of the secondary phase without further investigation of its character in details They explained it in several ways One assumed that this agent was a manifestation of latent spontaneous nystagmus caused by optokinetic stimulation, or assumed that it was a reflex phenomenon due to counter turning sensation Others explained it due to some central excitability like as optokinetic after nystagmus *In the author's opinion, the secondary phase does exist not only* in rotatory nystagmus, but also in both optokinetic and caloric nystagmus If it cannot be observed, it is because of extinction due to inhibitory factors such as visual inputs or unknown effects Moreover, it will be reasonable to assume that the secondary phase of nystagmus is organized by primitive tracts in the brain stem and is constantly influenced and controlled by the higher vestibular and optokinetic nystagmus tracts such as the cerebrum or cerebellum, considering our experimental results

1 The secondary phase of optokinetic nystagmus is always evoked by optokinetic stimuli where all vestibular stimulat on are excluded

2 Latency up to the occurrence of secondary phase and duration of secondary phase varies greatly in the same person even when the test is performed in the same manner by the same examiner

3 In rabbits extirpation of the bilateral cerebral or cerebellar hemispheres promote the occurrence of secondary phase

4 The higher the sensitivity of the vestibular and optokinetic systems, the higher the incidence of the secondary phase The fact that the secondary phase is liable to be evoked on the D P side is easily explained as a results of unbalance between the bilateral higher vestibular and optokinetic nystagmus tracts

Finally, the phenomenon of so-called " Optokinetic Nystagmus Firing " has not been found in the literature up to the present day It may be interpreted in several ways Bender 1956, studying optokinetic after-nystagmus in the monkey states that once rhythmical eye movements such as optokinetic nystagmus are set up, they continue of their own accord and become relatively independent of the original stimuli in regard to their frequency and duration Therefore, it is possible that the optokinetic stimuli create a corresponding repetitive, rhythmical discharge within the nervous system and once this activity is evoked it also becomes relatively autonomous but subject to the effects of visual stimuli The persistence of repetitive rhythmical activity within the nervous system is a well known phenomenon and has been explained in various ways Whether any of these explanations can be applied to this phenomenon is a subject for consideration An investigation of the anatomic and physiologic details of the brain stem will be necessary to answer this problem But, the presence of this phenomenon supplies us counter-evidence opposing the theory advanced by Barany and others that " Optokinetischer Nystagmus ist jedem bekannt durch die ruckartigen Augenbewegungen, die beim Blick

aus dem fahrenden Eisenbahnzug entstehen Neu auftauchende Teil der vorbeiziehenden Umwelt werden mit einer raschen Spahbewegung erfasst und dann mit langsamer Folgebewegung festgehalten, bis wieder ein neues Objekt den Blick auf sich zieht" More quantitative data, however, will be needed to substantiate it in the future

ZUSAMMENFASSUNG

Unter verschiedenen Bedingungen haben die Autoren aufgesucht die sekundare Phase des vestibularen und optokinetischen Nystagmus, die in normalen Menschen und Kaninchen hervorgerufen worden sind Die gewonnenen Resultate sind forgendes

1) Die sekundare Phase des Nystagmus kann leicht entlockt werden selbst durch schwache Reize, die klinisch gebraucht werden, sei es ein kalorischer Reize, sei es ein optokinetischer

2) Nach der Meinung der Autoren ist die sekundare Phase des Nystagmus organisiert durch die primitiven Bahnen im Hirnstamm und bestandig beeinflusst und beherrscht durch die hoheren vestibularen und optokinetischen Nystagmus Bahnen, wie das Grosshirn oder Kleinhirn

3) In Kaninchen haben die Autoren einige Falle erfahren in denen der optokinetische Nystagmus in Augenbewegungen transformiert wird, die an das sogenannte Nystagmus Klonus oder Feurung erinnern, welche in bezug auf die Frequenz des Nystagmus vollstandig von den orginaren Reizen unabhangig werden Das Vorhandensein dieser Erscheinungen reicht uns einen Beweis gegen die Baranysche Theorie dar uber die Genese des optokinetischen Nystagmus

REFERENCES

BÁRÁNY R, 1907 *Physiologie und Pathologie des Bogengangsapparates beim Menschen* Leipzig u Wien, Franz Deuticke

ASCHAN G and M BERGSTEDT 1955 The genesis of secondary nystagmus induced by vestibular stimuli *Acta Soc Med Upsal 60* 113

WAGEMANN W, 1958 Nystagmographische Beobachtung bei Kalorischen Dauerbelastung eines Bogengangsapparates *Arch Ohr usw Heilk 173* 346

BÖNINGHAUS H G 1953 Calorization und mehrphasiger Nystagmusablauf *Arch Ohr usw Heilk 164* 50

FISCHER M H und E WODAK, 1922 Experimentelle Untersuchungen uber Vestibularisreaktionen *Zschr Hals Nas u Ohrenheill 3* 198

FISCHER M H 1928 Die Regulationsfunctionen des menschlichen Labyrinthes und die Zusammenhang mit verwandte Funktionen *Ergebn der Physiol 27* 209

VEITS C 1931 Zur Drehprufung *Z Hals Nas u Ohrenheilk 29* 368

LANGE, C 1939 Untersuchungen uber den Ablauf der II postrotatorischen Reaktionen *Arch Ohr usw Heilk 146* 302

SCHMALZ G 1932 Physical phenomena occurring in semicircular canals during rotatory and thermic stimulation *Proc Roy Soc Med Sect Otol 25* 359

MITTERMAIER R 1938 Der Nystagmus postrotatorious II als klinische Untersuchungsmethode *Arch Ohr usw Heilk 144* 403

ASCHAN, G, 1955 The caloric test. *Acta Soc Med Upsal* 59 3
MITTERMAIER, R und G ROSSBERG 1956 Vestibularisuntersuchungen mit schwellen ahen Beschleunigungsreiz. *Arch Ohr usw Heilk*, 168 313
Van EGMOND A A J, J J GROEN and L. B W JONGKEES, 1952 The function of the vestibular organ New York.
THORNVAL A, 1936 Quelgues experiences sur la reaction calorique apres les injections dean froide et chaude *Acta Otolaryng*, 17 163
JUNG, R, 1953 *Handbuch der Inneren Medizin* 4 Aufl 5/1, Neurologie 1, s 1345
STAHLE, J, 1958 Electronystagmography in the caloric and rotatory tests *Acta Otolaryng*, *Suppl 137*
HAMERSMA, H, 1955 *The caloric test* Diss Amsterdam
McLAY, K, M F MADIGAN and F C ORMEROD, 1958 Electrical nystagmography and its use in the clinical investigation of vestibular function *J Laryng* 72 48
GRUTTNER, R 1939 Experimentelle Untersuchungen uber den optokinetischen Nystagmus *Zschr Sinnesphysiol* 68 1
MACKENSEN, G, 1958 Zur Theorie des optokinetischen Nystagmus *Klin Mbl Augenheilk* 132 767
KRIEGER, H P and M B BENDER 1956 Optokinetic afternystagmus in the monkey *EEG Clin. Neurophysiol*, 8 97

Asahimachi Niigata

ON THE FUNCTION OF THE OTOLITHORGANS

HIROSHI SASAKI MASATOSHI YAMAGATA, TETSUJIRO WATANABE
KEN ICHI OGINO MASAO ITO and SHUN ICHI OTAHARA
Yonago Japan

*From the Department of Oto Rhino Laryngology (Head Prof H Sasaki)
Tottori University Medical School Yonago*

Authors studied by several methods on the mechanism of the stimulation and effects of the otolithorgans of the animal and the human body The conclusions were as follows the otolithorgans were most effectively stimulated when the direction of acceleration was perpendicular to the macula The functions of saccule are believed to augment each other and have a rule in the formation of individual motion and position

INTRODUCTION

The relation between the linear accerelation and the otolithorgan has occupied the attention of many researchers but there are still quite different opinions concerning the mechanism of stimulation and its effects Authors applied to rabbits with the injured saccular macula, a simple harmonic moving apparatus the electromyograph and electronmicroscope for the study of the function of the otolithorgans

THE EXPERIMENTAL STUDIES OF THE SACCULE APPARATUS

A parallel swing apparatus was applied to the research of linear acceleration, however, the swing movement has such a limitation of use as to the constancy of width and frequency of movement Therefore, authors made a motor driven swing apparatus which ran on the linear rail By means of this apparatus swing movement arcs of 30 cm, 40 cm and 50 cm were able to be produced Acceleration at the end of the simple arc movement could be varied between 9 cm/sec² in the minimum and 625 cm sec in the maximum

METHOD

A rabbit was put on the apparatus and given a linear acceleration to the vertical saggital and frontal direction to the body axis

The kinetic reflex of M triceps brachii was then investigated by means of electromyograph This method has the advantage of making possible the objective observation of reflex due to stimulation quantitatively

arying length of linear movement The strength of stimulation could hanged stepwise, so that the variation of the reflex could be examined ematically

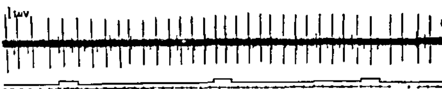


Fig 1 Electromyogram of tonic neuromu cular unit from M triceps brachii

RESULTS

The electromyograms of normal rabbits during the swing at the width 50 cm, with the linear acceleration of 9 cm/sec² in the saggital and ntal plane, show periodic changes in discharge intervals When the eleration is increased to the grade of 70 cm/sec or 100 cm/sec², a arkable kinetic reflex is produced During the acceleration to the tical direction with the rabbit bound in a lateral position (abnormal ition), the kinetic reaction was appeared at 35 cm/sec²

Rabbits with the bilateral total destruction of the labyrinths, howr, did not show any systematic reactions when linear accelerating nuli were applied Then a rabbit with the saccule damaged by opeion was compared with a subject operated in the similar method h the saccule left intact By gross observation the former had no ontaneous nystagmus, no head rotation and no influence in its kinetic lex, and the rabbit generally showed no quick reactions The rabbits od especially moving foreward with his head nodded up and down is phenomenon was remarkable immediately after destruction, but, after minutes, became obscure The rabbit was placed on the swing apratus, various linear accelerating stimuli were applied and the threshold reaction was surveied through recordings of electromyograph in the re legs

This experiment was done one day after the operation The rabbit th the damaged saccule showed a slightly increased threshold in comrison with that of a normal one During swinging in the saggital direcon, the threshold increased from 9 cm/sec to 35 cm/sec², which is not markably higher than the threshold of a normal This slight variaon from normal value is supporsed by authors to be due to the impaired nction of the total labyrinth rather than of the damaged saccule

During swinging in the frontal direction the degree of impaired reflex as somewhat greater than in the saggital direction The threshold ineased from 9 cm/sec to 70 cm/sec² The degree of impairment in this

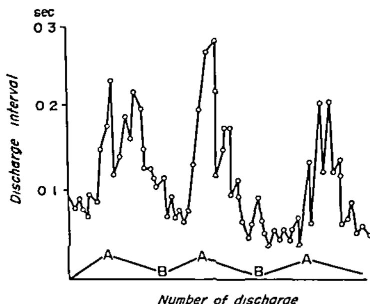


Fig 2 Interval diagram of a normal rabbit (the width of swing is 30 cm, A B period of swing is 2 7 sec, M triceps brachii)

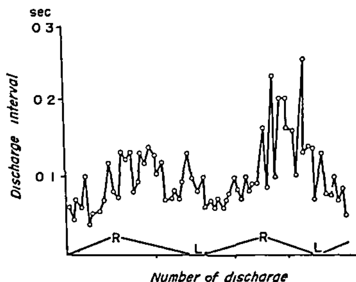


Fig 3 Interval diagram of a normal rabbit by the swing in the frontal plane (R right, L-left)

animal was greater than in a control Subsequently during swing in the vertical, the threshold of reaction in the damaged animal increased from 9 cm/sec² to 70 cm/sec² However, the apparatus in this experiment was not adequate to investigate this response If an up-to down apparatus was available more accurate values of threshold of the reflex should be able to obtained Since the above results are not conclusive, it would be inappropriate to draw any conclusion on the function of the otolithorgans

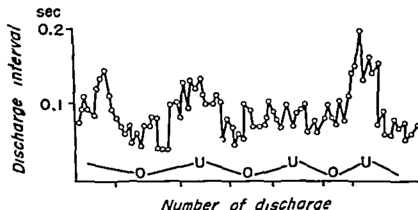


Fig 4 Interval diagram of a normal rabbit during swing to vertical direction (O up U-down)

However, following suggestion could be made,

1) The saccule reacts continuously, however, its function is not decisive to the movement and posture of the body

2) The compensation occures early following damage to the saccule, therefore no remarkable abnormality can be observed in movement and posture of the body

3) Even when no apparent change of reflex is observed, an increased threshold of the kinetic reflex can be proved by electromyographic analysis of the extremitus muscle

4) In rabbits the linear kinetic reflex is influenced by the saccular macula during movement from side to side

5) The saccular macula reacts only when the linear accelerating stimulus is perpendicular to the plane of the macula

The suggestion mentioned above indicates two new ideas Firstly, the electromyogram can be effectively used for measurement of the function of the otolithorgan and secondly, it is possible to stimulate each otolithorgan selectively

However these procedures must be carried out carefully under the specific conditions

Ashcroft and Hallpike (1934) recorded action currents in response to tunig fork vibration from the saccular nerve of a frog Experiment were carried out to investigate acoustic function of the saccule and electric potential changes in response to the sounds were recorded by authors

The findings of Ashcroft and Hallpike (1934) were confirmed by authors Hand clapping human voice and pure tones from audiometer were used as sound stimuli

Using pure tones the threshold of stimulating sound which caused potential changes was measured The following data were obtained

1) Between 45 and 50 dB in 125 cps

2) Between 55 and 60 dB in 250 and 500 cps
3) Between 75 and 85 dB in 1000 cps
4) No response was obtained at 75 dB in 1500 cps

On the basis of our experimental results, it is noted that the threshold becomes lower in proportion to the decrease of frequencies of the sound stimuli such as vibration, hand clapping and pure tones and that no response could be obtained at 75 dB in 1500 cps

It can be assumed, therefore, that the saccule of a frog has some function in acoustic perception

THE EXPERIMENTAL STUDIES OF THE UTRICLE

METHOD

Many problems are encountered in the study of the function of the otoliths In order to observe the otolithic response selectively one of the most difficult problems is to eliminate sensations of other organs such as eyes, skin and joints, which are also influenced by linear acceleration In addition, authors stress the fact that both otolithorgans are augmented and suppressed each other The best method concerning this matter is to pick up centrifugal signal from the peripheral receptive apparatus as in the study of the ray by Loewenstein (1950)

However, as the action current from the nerve of the warm blooded animals is very unstable, it is neccessary to control the room temperature and humidity carefully during the experiment

After many failures, the procedure by which access to the basal turn of the cochlea through neck incision is possible is found by authors A needle electrode of about 10μ in diameter was inserted into the utricular nerve in the partition between the utricle and the saccule

The action current from the utricular nerve of a rabbit was mostly biphasic or monophasic and its amplitude was between 50 to 200μ volts

When the rabbit was placed on its back, spontaneous discharge from the utricular nerve was recorded sporadically The difference of spikes seemed to be due to potentials picked up from several nerves with one electrode

The experiment should be completed within 5 minutes after operation since the spontaneous discharge gradually decreased and disappeared within 30 minutes even under the most carefully controlled conditions

RESULTS

Nine rabbits which were successfully operated and of which the action current from the utricular nerve were placed on the swing apparatus

The changes of discharge interval were investigated during a single swing of width of 50 cm and of 2 5 sec in a cycle

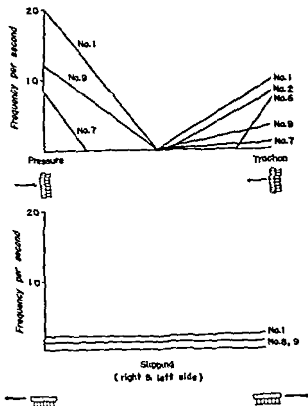


Fig 5 Relationship between linear acceleration to the utricular macula and discharge frequency of the action current from the utricular nerve

Three different kinds of phenomenon were observed from relationship between the direction of linear acceleration and the position of the utricular macula These are the pressure, traction and displacement of the otolith to the sensory epithelium

The average of discharge frequency was suggested to have some significance in spite of large individual differences The discharge was frequent when the macula was stimulated by pressure, and less frequent when stimulated by traction and sporadic continuous discharge was recorded when stimulated by displacement to the lateral side

Though different values under the same circumstances were observed, it could be concluded that the utricular macula has two modes of reaction

It is interesting to know whether the otolith crystals respond to acceleration of gravity or not We fixed the rabbits on an experimental table allowing them to change their positions (nose-up or nose-down) and then the table was rotated around the horizontal axis The rabbits used in this experiments were freshly operated and were not one to be used in previous experiment. In order to obtain stable continuous discharge, special attentions were paied to the experimental technique.

2) Between 55 and 60 dB in 250 and 500 cps
3) Between 75 and 85 dB in 1000 cps
4) No response was obtained at 75 dB in 1500 cps

On the basis of our experimental results, it is noted that the threshold becomes lower in proportion to the decrease of frequencies of the sound stimuli such as vibration, hand clapping and pure tones and that no response could be obtained at 75 dB in 1500 cps

It can be assumed, therefore, that the saccule of a frog has some function in acoustic perception

THE EXPERIMENTAL STUDIES OF THE UTRICLE

METHOD

Many problems are encountered in the study of the function of the otoliths In order to observe the otolithic response selectively one of the most difficult problems is to eliminate sensations of other organs such as eyes, skin and joints, which are also influenced by linear acceleration In addition, authors stress the fact that both otolithorgans are augmented and suppressed each other The best method concerning this matter is to pick up centrifugal signal from the peripheral receptive apparatus as in the study of the ray by Loewenstein (1950)

However, as the action current from the nerve of the warm blooded animals is very unstable, it is neccessary to control the room temperature and humidity carefully during the experiment

After many failures, the procedure by which access to the basal turn of the cochlea through neck incision is possible is found by authors A needle electrode of about 10μ in diameter was inserted into the utricular nerve in the partition between the utricle and the saccule

The action current from the utricular nerve of a rabbit was mostly biphasic or monophasic and its amplitude was between 50 to 200μ volts

When the rabbit was placed on its back, spontaneous discharge from the utricular nerve was recorded sporadically The difference of spikes seemed to be due to potentials picked up from several nerves with one electrode

The experiment should be completed within 5 minutes after operation since the spontaneous discharge gradually decreased and disappeared within 30 minutes even under the most carefully controlled conditions

RESULTS

Nine rabbits which were successfully operated and of which the action current from the utricular nerve were placed on the swing apparatus

The changes of discharge interval were investigated during a single swing of width of 50 cm and of 2 5 sec in a cycle

charge were picked up from several nerve fibers Though recording of a response from one single nerve fiber was tried, it was failed because of technical difficulty

In general, the greatest response was observed by pressure and traction Some responses by backward displacement were observed in several cases It is reason why the surface of the utricular macula was not completely horizontal in normal head position but the fore portion was planted slightly upward Therefore, by backward displacement of the utricular macula the elements of traction and pressure existed

Our interpretation of the function of the otolithorgan as based on the results of several fundamental experiments is as follows,

Firstly, the saccule is a receptor of the reflex which is very sensitive to linear acceleration to direction to perpendicular to its surface Secondly, the saccular macula does not play the leading part in response to the acceleration to the right and left

Obtaining the action potential directly from the saccular nerve, it was noted that the linear acceleration was effective as a stimulus when the direction of the acceleration was in perpendicular to the saccular macula Therefore it was understood that the acceleration to the three directions that is to say, right and left, up and down, for and backward, would be perceived properly by the body bacause of the special structure of the otolithorgan

Authors were interested in two kinds of sensory cells demonstrated in the utricular macula by Smith (1959) while studying complex function of the otolithorgans By using an electron microscope, authors successed to differentiate two types of cells which were morphologically different and had different innervation However, at present the relationship between the function of these cells of two types and function of two modes of the otolithorgan was not understood by authors

THE CLINICAL EXPERIMENT

Authors investigated the threshold of the kinetic reflex and sensation to the linear acceleration in the human body by a similar method to the above mentioned basic experiments

The most difficult problem in the clinical experiment was how to detect a suitable indicator In addition to this both otolithorgans are practically influenced each other in the human body Moreover, the surfaces of the otolithic maculas are not simply flat and the planes of both maculas are not truely at right angles The linear accelerations of several degrees were applied stepwise and selectively to the macula, and then the spike discharge from the anterior tibial muscle were investigated by electromyogram



Fig 7 Electron microscopic picture

H Hair
M_1, M_2 Mitochondria
Nu_1, Nu_2 Nucleus
N_1 Nerve endings
C Cuticula
S Supporting cell
MV Microvilli
R Reticulum lamina
HC I Flask shaped cell
HC II Oval shaped cell

It is much more difficult in a human body than in an other animal that the linear kinetic reflex is picked up, and it has been generally said that the kinetic reflex in a human body could not be observed But there is no doubt that this reflex exists in a normal human body and the existens of this reflex has been proved by electromyography

Authors investigated this reflex to the linear acceleration of various degrees and found that the threshold in this reflex was approximately 9 cm sec This threshold is changed by physical training and in cases of athletes apparent changes in discharge interval were demonstrated This reflex is easily demonstrated when the direction of acceleration is in the plane of longitudinal axis of the body, which is the direction at right angles to the utricular macula The reflex is less easily demonstrated when the direction of acceleration is in the plane of frontal axis of the body or perpendicular to the saccular macula

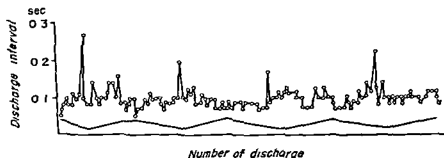


Fig 8 A case of sportsman, vertical direction, the swing period of 10 sec, (M tibialis ant. sin)

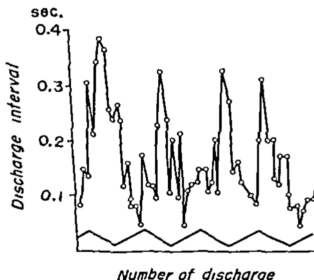


Fig 9 A case of hyperfunction of vestibular apparatus, vertical direction, the swing period of 2 7 sec (M tibialis ant dext)

Authors observed this reflex while the subject's head was inclined rward or tip backward, thereby changing the relationship of the ricular macula to the direction of acceleration Although the influence the neck reflex can not be completely disregarded, authors believe that langes of the direction of acceleration to the macula give much more ifluence to the reflex than the neck reflex gives

No difference of response was noted between in normal head position nd in forward flexion of 30 degrees of the head

The decrease of the response was noted in the head position of backard flexion of 60 degrees Similar investigation was carried out during cceleration in the frontal axis of the body No difference of response as noted between in the lateral head position of 45 degrees and in a ormal position

These results possibly suggest the existance of interaction between both otoliths

In the cases of athletes fluctuation of frequency of discharge was not remarkably great but extremely regular even during stimulation of acceleration of 9 cm/sec^2

The frequency of discharge changed sensitively in response to change of acceleration In the cases of hyper function of the labylinth this reflex responded to the change of acceleration very well but changes in muscle tonus were very large but unstable

In the cases of the hypo function of the labyrinth, only a slight response of the reflex was detected under the acceleration of 140 cm/sec^2 and it was difficult to detect the response to the acceleration of 35 cm/sec^2 In the subjects, whose vestibular apparatus was non functioning the re sponse was not detected under the acceleration of neither 35 cm/sec nor 140 cm/sec^2, and the discharge interval was irregular and showed no periodic fluctuation

Last of all, using our apparatus, authors examined the linear kinetic sensation *Many receptors are involved in this sensation and it is difficult* to decide the true sensitivity of the otolithorgan

The subject was placed in the supine position with eyes covered on a thick soft blanket and the apparatus was set in motion The stimulus load was a cycle of 10 seconds and amplitude of 50 cm and the maximum acceleration was 9 cm/sec^2 at both ends of arc

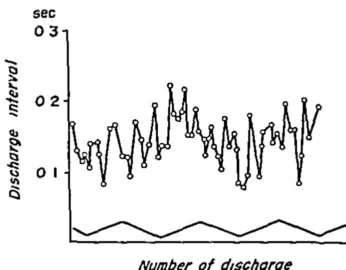


Fig 10 In a case of hypofunction of bilateral vestibular apparatus stimulus in vertical direction with a swing period of 2 7 sec (M tibalis anterior)

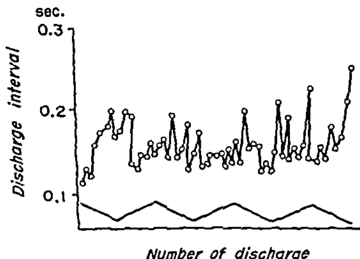


Fig 11 A case of the bilateral labyrinths are destroyed, stimulus in vertical direction with a swing period of 2 7 sec (M tibialis anterior)

Healthy adults showed distinctly individual differences in discriminating the direction of acceleration, but they could discriminate accurutely with the acceleration of 9 cm/sec² The threshold of sensation increased to the acceleration of 35 cm/sec in cases with the vestibular impairment

DISCUSSION AND CONCLUSION

At present, there is no doubt that the otolithorgans respond to the linear acceleration However, the role of this reaction in mammals, particularly in human bodies, has not been clearly understood Authors applied the knowledge gained from the animal experiment to human beeings in order to utilize this idea clinically

Although there were many difficulties, it could be ascertained that the reflex and the sensation of the mortion in response to the gradual changes of the acceleration were influenced by the otolithorgans However, it seems doubtful whether the otolithorgan alone plays a decisive role or not The compensation is supposed to take place in early stage and the remaining function is seemed to be influenced by the semicircular canal

The otolithorgans were most effectively stimulated when the direction of acceleration was perpendicular to the macula

The saccule and utricle are believed to augment each other and have a rule in the formation of kinetic and positional reflex The otolithorgans respond to both pressure and traction, therefore, it is assumed that the acceleration of the linear movement and the acceleration of gravity is

perceived in the peripheral organ but all information from the peripheral organs may be integrated in the central nervous system and the proper response is produced by this way

ZUSAMMENFASSUNG

Unsere Autoren stimulierten die Otolithorganen mit der linearen Beschleunigungen zur einigen Richtungen, um ihre Empfindungsmechanismus klinisch sowohl und tierexperimentell aufzuklaren

Es wurde konstatiert, dass die Otolithorgane die starkeste Reaktion zeigten, wenn die Richtung der linear Beschleunigung waren perpendikular gegen die macular Flache des Sacculus und des Utriculus

Bei der Gestaltung der Bewegung und Stellung eines Individums spielen diese beiden Organe, die wirkend miteinander, eine grosse Rolle

REFERENCES

ADRIAN, E D, 1942 Discharges from vestibular receptors in the cat *J Physiol*, *101*, 389

ASHCROFT D W, & HALLPIKE C S, 1934 On the function of the saccule *J Laryng* 49, 450

BENJAMINS C E, 1935 Die Funktion des Sacculus *Acta oto laryng* *22* 338

DE KLEIJN, A und VERSTEEGH, C, 1935 Naheres uber die Auslosungsstelle der Labyrinthreflexe im peripheren Labyrinth *Acta oto laryng*, *22* 327

GERNANDT, B, 1945 Response of mammalian vestibular neuron to horizontal rotation and caloric stimulation *J Physiol* *12*, 173

HAAS E und MATZKER J, 1958 Über der Einfluss der Linearbeschleunigung auf die Entstehung von Vestibularisschwindel *Z fur Laryng* *37*, 17

JONGKEES L, B, W, 1950 On the function of saccule *Acta oto laryng* *38* 18

LOEWENSTEIN, O, & ROBERTS T D M 1950 The equilibrium function of the otolith organs of the Thornback Ray (Raja Clavata) *J Physiol* *110* 392

MAGNUS, R, 1925 *Korperstellung* Berlin, Julius Springer

MC NALLY W J, 1927 Function des runden Fensters beim Kaninchen *Acta oto laryng* *51* 40

MC NALLY W J, 1947 The physiology of the vestibular mechanism in relation to vertigo *Ann Otol etc St Louis*, *56* 514

OGINO K 1959 Experimental study on utricular function by inducing action current of utricular nerve *Otologica Fukuoka*, *6* 72

SASAKI H 1960 On the function of the otoliths *Jap Jour Otol Tokyo* *63* 624

SASAKI H & MIYATA J, 1955 Experimental studies on otoliths *Z Laryng Stuttgart* *34* 740

SMITH C A 1956 Microscopic structure of the utricule *Ann Otol etc St Louis* *65* 450

TOKIZANE, T MURAO M OGATA T and KONDO T 1951 Electromyographic studies on tonic neck lumbar and labyrinthine reflexes in normal persons *Jap J Physiol* *2* 130

VAN EGMOND A A J GROEN J J, and JONGKEES L B W, 1952 Function of the vestibular organ *Pract Oto Rhino Laryng*, *54* 101

VERSTEEGH, C, 1927. On the function of the vestibular organs *Acta oto-laryng*, *11*, 393

WATANABE, T, 1959. Experimental studies on the kinetic reflex and the sensation to the linear Acceleration in the human bodies *Jap Jour Otol Tokyo*, *62*, 99

YAMAGATA, M, 1958 Experimental studies on the function of saccule. *Jap Jour Otol. Tokyo*, *61*, 1931.

Nishimachi, Yonago

ÜBER DIE LATENZZEIT DES KALORISCHEN NYSTAGMUS

HATSUO INO und KEN OKAMOTO
Tokio, Japan

Aus der Hals Nasen Ohrenklinik, Tokyo 2 Nationales Krankenhaus (Director Dr H Ino) Tokio

Die Verfasser haben die Entstetehung der Latenzzeit des kalorischen Nystagmus ausfuhrlich untersucht, wie alle Teile des Vestibularissystem sich dabei beteiligen, und einige folgende Befunde berichtet.

1) Das Warmeleitungssystem (ausserer Gehorgang, Trommelfell, Schlafenbein) spielt eine grosse Rolle

2) Latenzzeit und Nystamusdauer verlaufen in einer bestimmten Korrelation

3) Die echte Latenzzeit hat den wichtigen diagnostischen Wert

EINLEITUNG

Mit dem Wort "die Latenzzeit des kalorischen Nystagmus" meint man die Zeitdauer vom Beginn der Einspulung bis zum Auftreten der ersten langsamen Phase des Nystagmus

Diese Latenzzeit ist abhangig von der anatomischen Konstruktion des Schlafenbeins und ist individuell verschieden Anderseits wird sie leicht durch nonlabyrinthare Faktoren beeinflusst Aus diesen Grunden hatte die Latenzzeit bisher im allgemeinen keine grosse Aufmerksamkeit gefunden Wenn man jedoch manche klinischen Falle ausfuhrlich erforscht, bemerkt man, dass der diagnostische Wert der Latenzzeit nicht gering ist

Wodack (1952) und Ino (1955) haben aus ihren klinischen Erfahrungen darauf hingewiesen, dass die Latenzzeit die Diagnose bedeutend unterstutzen kann Gegenuber den Autoren, die glauben, dass sie nicht durch die Veranderung des peripheren Labyrinths, sondern eher durch die anatomische Struktur des Schlafenbeins bzw die Durchblutung beeinflusst wird, und der Latenzzeit keinen grossen Wert zuerkennen, betont Wodack, dass sie durch irgendeine Veranderung des peripheren Labyrinths hervorgerufen wird Aus ahnlichen Gesichtspunkten hat Ino theoretisch eine Bestimmungsmethode der sogenannten "echten Latenzzeit", in der der anatomische Faktor ausgeschlossen ist, erfunden und den "Z-Test" vorgeschlagen mit dem man neben "Z" — die echte Latenzzeit — auch die Nystagmusdauer bestimmen kann Er versucht, aus der Beziehung beider Werte den Herd lokal zu diagnostizieren

Es ist uber den Entstehungsmechanismus des karolischen Nystagmus schon bekannt, dass die Temperaturveranderung durch Spulung des ausseren Gehorgangs zuerst ins Labyrinth gelangt und die Endolymphstromung hervorruft Die infolgedessen hervorgerufene Ablenkung des Cupulas lasst den afferenten Impuls durch den Nervus vestibularis, Nucleus vestibularis, Fasciculus long post und Augenmuskelkern zum Augenmuskel gelangen Danach tritt die langsame Phase des Nystagmus erst in Erscheinung

Weiter ist es bekannt, dass die obengeordnete Zentren wie das Grosshirn bzw Kleinhirn gegenuber dem primaren Nystagmusreflexbogen eine kontrollierende Rolle spielen Es kann daraus angenommen werden, dass, wenn dieses Vestibularissystem irgendwo gestort wird, eine Veranderung auch in der Latenzzeit in Erscheinung tritt Auf dieser Ansicht beruhend haben wir die Faktoren, die sich zwischen der Peripherie und dem Zentrum finden, kritisch untersucht, um den Entstehungsmechanismus der Latenzzeit zu erforschen

Die Latenzzeit kann per Abb 1 schematisch analysiert werden „a" + „b" + „c" stellen die Latenzzeit dar, die bisher ublich bestimmt wurde „a" zeigt die Zeit, von der Spulungsbeginn bis zum Eintreten der Temperaturveranderung durch das Schlafenbein bis in die Ampulla „b" zeigt die Zeit, die von dem Eintreten der Temperaturveranderung in der Ampulla bis zum Auftreten des Impulses im Nervus vestibularis gebraucht wird „b" wird weiter in x, y und z eingeteilt „x", „y" und „z"

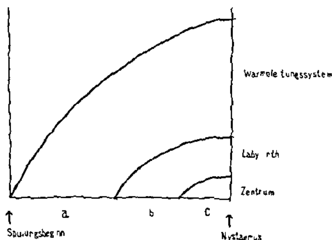


Abb 1 Analyse der Latenzzeit

stellen im einzelnen die Zeit dar, die zum Eintreten der Temperaturveranderung von der Ampulla bis zum perilymphatischen Raum gebraucht wurde die zum Eintreten der Temperaturveranderung aus dem perilymphatischen Raum in den endolymphatischen Raum gebraucht wurde und die Zeit die vom Eintreten der Temperaturveranderung in der Endolym

Zur Bestimmung der Latenzzeit wurde ausschliesslich der Braunsche Oscillograph und zur Beobachtung der Nystagmusdauer der Penoscillograph angewandt Zum Hervorrufen des Nystagmus wurde eine Leitungs-

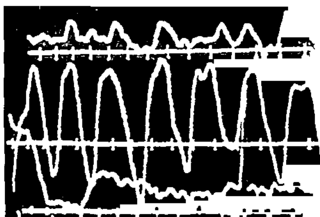


Photo 2 Registrierung durch Braunsche Oscillograph

elektrode in den inneren und ausseren Augenwinkel auf einer Seite subcutan hineingestochen Die Indifferenzelektrode wurden subcutan in die Stirn gelegt

Um die Frage der Sensibilitat des ausseren Gehorgangs zuerst zu erforschen, wurde 1 %ige Novokainlosung in den ausseren Gehorgang des Kaninchens injiziert Vor und nach der Injektion wurden Testspulungen durchgefuhrt, um den Verlauf der Latenzzeit zu beobachten Aber bei diesem Experiment durch Injektion der Novokainlosung verengt sich der von Natur aus enge aussere Gehorgang weiter Infolgedessen kann das eingespulte Wasser nicht mehr in genügender Menge zum Trommelfell gelangen und die Latenzzeit wird nur sehr ungenau bestimmt Daruber hinaus tritt selbst der Nystagmus oft nicht mehr in Erscheinung Daher haben wir diese Methode nicht verwandt

Wir haben deshalb den ausseren Gehorgang an der Grenze von dem knochernen ausseren Gehorgang ausgeschaltet und direkt zum blossgelegten Trommelfell eingespult Die Einspulung wurde unter derselben Bedingungen wie vor der Operation durchgefuhrt So wurde der Verlauf der Latenzzeit und Nystagmusdauer untersucht Durch diese Methode kann das Problem der Sensibilietat bzw Temperaturveranderung des ausseren Gehorgangs ganz ausgeschlossen werden

Danach wurde das Trommelfell ausgeschaltet und der Attik mit einem Bohrer erweitert damit die Ampulle des horizontalen Bogengangs direkt gesehen werden konnte Dann wurde Wasser unter derselben Bedingungen eingespult und die Latenzzeit und Nystagmusdauer bestimmt Die Ergebnisse werden in die Abb 2 aufgefuhrt

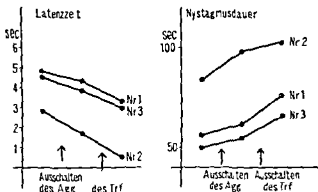


Abb 2 Veranderungen der Latenzzeit und Nystagmusdauer nach Ausschalten des ausseren Gehorgangs und Trommelfells

Wie aus Abb 2 ersichtlich ist, befinden sich bei der Latenzzeit bzw Nystagmusdauer ziemlich grosse individuelle Differenzen je nach den Tieren Die Latenzzeit zeigt bemerkenswerte Schwankungen bei den Tieren, sei es Wasser durch den ausseren Gehorgang, direkt auf das Trommelfell nach dem Ausschalten des ausseren Gehorgangs oder direkt auf das Labyrinth nach dem Ausschalten des Trommelfells eingespult wurde

Diese Tatsache weist auf die grosse Gefahr hin, die Funktion des Labyrinths danach zu beurteilen, und die erhaltene Latenzzeit mit denen anderer Tiere zu vergleichen Diese Tatsache steht mit der bisherigen Ansicht im Einklang, die behauptet, dass die Latenzzeit je nach der anatomischen Differenz des Schlafenbeins verschieden ist

Auch aus der Tatsache, dass die Latenzzeit eine betrachtliche individuelle Differenz aufweist, wenn auch in dem Fall, dabei Wasser direkt auf den Bogengang eingespult wurde, kann man schliessen, dass das Warmeleitungssystem bei der Untersuchung des kalorischen Nystagmus eine auffallende Rolle spielt und es hier eine grosse individuelle Differenz gibt

Aber wenn die Beobachtung auf dasselbe Tier beschrankt wurde, verursachte das Ausschalten des ausseren Gehorgangs eine Verkurzung der Latenzzeit und eine Verlängerung der Nystagmusdauer Wenn das Trommelfell ausgeschaltet und Wasser direkt auf den Bogengang eingespult wurde verkurzte sich Latenzzeit und verlangerte sich die Nystagmusdauer weiter Noch dazu betragt die Verkurzung der Latenzzeit bei jedem Fall ca 10 % nach dem Ausschalten des ausseren Gehorgangs und ca 20 nach dem Ausschalten des Trommelfells Aus diesen Ergebnissen kann man schliessen, dass das Warmeleitungssystem fur ca 30% der Latenzzeit verantwortlich ist

Dann haben wir die Temperatur des ausseren Gehorgangs verandert, um zu untersuchen, wie die Latenzzeit dann verlauft. Am Kaninchen

wurden vorher Testspulungen durchgefuhrt und die Latenzzeit und Nystagmusdauer bestimmt Nachdem seine Ohrmuschel auf einer Seite mit einem gedampften Handtuch ca 5 min lang gewarmt wurde, wurde die Latenzzeit bestimmt Dann wurde die Ohrmuschel mit einem eisgekuhlten Handtuch abgekuhlt und danach wurde die Latenzzeit bestimmt Wir haben so untersucht, wie die Latenzzeit nach der Spulung des kalten bzw warmen Wasser sich durch Erwarmung bzw Abkuhlung der Ohrmuschel verandert Wie aus Abb 3 ersichtlich ist, verlangerte sich die Latenzzeit — wenn kaltes Wasser eingespult wurde — durch Erwarmung der Ohrmuschel und verkurzte sich durch Abkuhlung der Ohrmuschel Umgekehrt verlief sie nach dem warmen Reiz, d h verkürzte sie sich durch Erwarmung der Ohrmuschel und verlangerte sich durch Abkuhlung *Nach einer langen Pause wurde dasselbe Experiment durchgefuhrt* Dieselbe Ergebnisse wurden beobachtet Dann wurde der aussere Gehor-

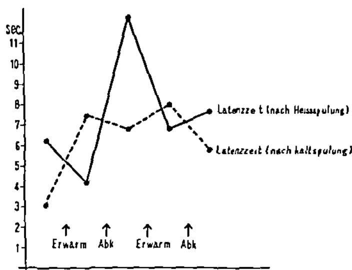


Abb 3 Verlauf der Latenzzeit durch Erwarmung bzw Abkuhlung der Ohrmuschel

gang an der Grenze zum knochernen ausseren Gehorgang fast ganz ausgeschaltet Er war durch einen kleinen Teil des weichen Gewebe noch mit dem knochernen ausseren Gehorgang verbunden In diesem Zustand wurde die Ohrmuschel erwarmt und abgekuhlt. Wie aus der Abb 4 ersichtlich, veranderte sich die Latenzzeit durch Erwarmung bzw Abkuhlung der Ohrmuschel gar nicht Sie halt beinahe einen bestimmten Wert ein Aus diesen Ergebnissen ist zu verstehen, dass die Latenzzeit durch Temperaturveranderung des ausseren Gehorgangs betrachtlich beeinflusst wird Deswegen scheint uns, dass, je grosser die Temperaturdifferenz zwischen dem ausseren Gehorgang und dem eingespulten Wasser ist desto langer wird die Latenzzeit und je kleiner sie ist, desto kürzer wird die Latenzzeit

Es gibt eine Ansicht, dass je grosser die Temperaturdifferenz zwischen dem Labyrinth und eingespulten Wasser ist, je starker fliesst die Endolymphe und ruft den Nystagmus leicht hervor Dagegen scheint die Tatsache, dass je grosser die Temperaturdifferenz zwischen eingespultem Wasser und dem ausseren Gehorgang ist, desto langer die Latenzzeit wird, zu bedeuten, dass je naher die Temperatur des Gehorgangs zu dem ihm eingespulten Wasser ist, desto schneller die Temperaturveranderung zum Labyrinth fortgepflanzt wird und so die Latenzzeit verkurzt

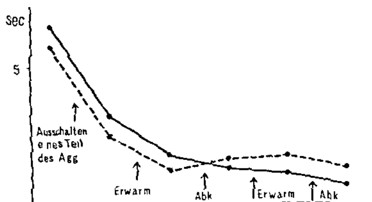


Abb 4 Verlauf der Latenzzeit durch Erwarmung bzw Abkühlung der Ohrmuschel nach Ausschalten des ausseren Gehorgangs

Da wir schon festgestellt haben, dass die Latenzzeit von der Temperatur des Gehorgangs sehr abhangig ist, haben wir dann Wasser direkt in das Labyrinth eingespult, um den Einfluss des ausseren Gehorgangs auszuschliessen Dabei ist es wichtig, die Temperatur des Labyrinths vorher genau zu bestimmen Dazu wurde die Temperatur der Ampulla des horizontalen Bogengangs des Kaninchens nach vorheriger Ausschaltung des ausseren Gehorgangs und des Trommelfells unter direkter Sicht mit dem Thermister genau bestimmt Danach wurde 1 cm³ von 1°C Wasser auf die Knochenwand des Labyrinths in Abstanden von 5 min eingespult und anschliessend daran wurden die Latenzzeit und Nystagmusdauer bestimmt Tab 1 stellt die Ergebnisse dar. Wie aus der Tabelle ersicht-

Tab 1 Korrelation zwischen der Temperatur des Labyrinths Latenzzeit und Nystagmusdauer

	1	2	3	4	5	6	7	8 Mal
Latenzzeit	1.1	1.3	1.2	1.6	1.0	0.7	1.0	1.1 Sec
Nystagmusdauer	70	72	76	86	72	83	80	85 Sec
Temperatur des Labyrinths	38.6	38.1	38.1	37.5	37.8	37.4	37.2	37.1 °C

lich, existiert keine bestimmte Korrelation zwischen der Temperatur des Labyrinths, Latenzzeit und Nystagmusdauer

Dann haben wir kaltes (1°C 1 cm³) und warmes (55°C 1 cm³) Wasser wiederholt eingespult, nachdem die Temperatur der Knochenwand des Labyrinths auf einer bestimmten Hohe bestandig geworden war, und die Korrelation untersucht Die Ergebnisse wurden in Tab 2 aufgefuhrt

Tab 2 Latenzzeit und Nystagmusdauer, die bei einer bestimmten Temperatur des Labyrinths bestimmt wurden

	1	2	3	4 Mal
Latenzzeit	1 0	1 0	0 9	0 9 sec
Nystagmusdauer	57	60	58	60 sec
Temperatur des Labyrinth	37.2 °C			

(1°C, 1 cm³—Spulung)

	1	2	3	4 Mal
Latenzzeit	0 9	1 0	1.0	0 9 sec
Nystagmusdauer	57	56	51	55 sec
Temperatur des Labyrinth	38 4 °C			

(55°C, 1 cm³—Spulung)

Es ist klar, dass die Latenzzeit und Nystagmusdauer stets fast bestandig sind, wenn die Spulung unter bestimmten Bedingungen — nachdem die Temperatur des Labyrinths eine fast bestimmte Hohe erreicht hat — durchgefuhrt wird

Aus obigen Tatsachen wurde festgestellt, dass obwohl die Ohrmuschel bzw der aussere Gehorgang uber das Warmeleitungssystem die Latenzzeit bedeutend beeinflussen, die Latenzzeit des normalen Kaninchens stets konstant ist, wenn Wasser nach der Stabilisierung der Temperatur des Labyrinths eingespult wurde

B) *Fragen des Labyrinths*

Obwohl manche problematische Frage noch im Entstehungsmechanismus des kalorischen Nystagmus liegt, ist heute die sogenannte Endolymphstromungstheorie die von Barany (1906) vorgeschlagen wurde, allgemein anerkannt

Seine Theorie wurde von manchen Autoren nachuntersucht, wie Steinhausen (1931 Beobachtung am Hecht), Dohlmann (1925 am Haifisch) und Maier Lion (1921 Beobachtung an der Taube) Sie haben die Ablenkung der Cupula festgestellt und unterstutzen die Endolymphstromungstheorie Anderseits nehmen Schmaltz (1932) und Kono (1951) nach ihren Temperaturbestimmungen an, dass die Temperaturdifferenz

zwischen der Ampulla und dem Schenkel den Fluss der Endolymphe hervorrufen

Wenn man die Endolymphstromungstheorie anerkennt, ist anzunehmen, dass die Latenzzeit durch irgend eine Veranderung des peripheren Labyrinths — sei es die Endolymphe od die Cupula — betrachtlich beeinflusst wird

Daher haben wir das Labyrinth experimentell verandert und untersucht, wie sich die Latenzzeit verandert

Zuerst wurden Kaninchen waagerecht fixiert und Wasser eingespult Die Kopflage, bei der kein Nystagmus in Erscheinung tritt, wurde fur die Lage, bei der der horizontale Bogengang horizontal liegt, angesehen Wasser wurde bei Kopflagen, bei denen der Kopf 30°, 60° bzw 90° zur Ruckenseite geneigt war, eingespult und die Latenzzeit wurde bestimmt Dabei wurden normale Kaninchen sowie Kaninchen mit Ausschaltung des ausseren Gehorgangs und des Trommelfells fur die Untersuchung verwandt Bei letzteren wurde Wasser direkt auf den Bogengang eingespult Abb 5 stellt den Vergleich der Ergebnisse dar Je starker der Kopf gebeugt wurde, desto kurzer wurde die Latenzzeit und langer die Nystagmusdauer

Es ist daher zu verstehen, dass es bei der Bestimmung der Latenzzeit des kalorischen Nystagmus notig ist, die Kopflage stets genau konstant

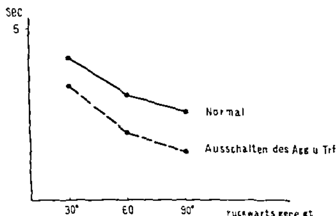


Abb 5 Die Kopflage und die Latenzzeit.

zu halten Dann haben wir beobachtet, wie sich die Latenzzeit bei irgend einer pathologischen Veranderung des Labyrinths andert Die Bulla eines Ohrs wurde aufgeschnitten Kokain-Alkohol Gemisch in gleicher Menge wurde eingespult und 5 min lang — bis zum Verschwinden des Spontannystagmus nach der gesunden Seite — wurde liegen gelassen Danach wurde die Latenzzeit bestimmt und die Ergebnisse mit denen vor der Operation verglichen Durch die Untersuchungen im Anschluss an das Verschwinden des Spontannystagmus wurde festgestellt, dass sich bei

einem Teil der Tiere die Latenzzeit und Nystagmusdauer von denen vor der Operation kaum unterschieden, und bei anderen Kaninchen, bei denen CP (canal paresis) auf der Wasser eingespulten Seite in Erscheinung trat, die Latenzzeit sich nach der Operation verlangert Dann haben wir die Latenzzeit auf der Seite, wo der horizontale Bogengang aufgeschnitten wurde, untersucht Wenn ein kleines Loch mit einem Bohrer an dem hinteren Teil des horizontalen Bogengangs gebohrt wurde, ist ausser dem geringen Abfluss der Lymphe ein Spontannystagmus nach der kranken Seite zu beobachten Nach einer Weile verschwand dieser Spontannystagmus Wenn eine Testspulung danach durchgeführt wurde, trat CP bei einem Teil von Kaninchen in Erscheinung Bei denen verlangerte sich die Latenzzeit auf der CP Seite

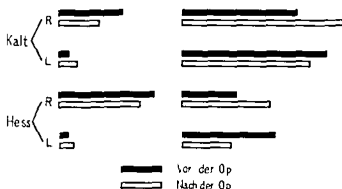


Abb 6 Veranderungen der Latenzzeit nach der Einspritzung der Kokain Alkohol Losung in die Bulla

Über Entstehung der Latenzzeit des kalorischen Nystagmus ist anzunehmen, dass dabei nicht nur das Bogengangssystem, sondern auch das Otolithensystem irgend eine Rolle spielt Ohwada (1960) hat experimentell den Sacculus bzw den Utriculus des Kaninchens einzeln zerstort und den Einfluss auf den Nystagmus erforscht Er wies in seinen Untersuchungen darauf hin, dass es moglich sei, dass der Befund der sogenannten "DP" (directional preponderance), der beim Meniereschen Syndrom in Erscheinung tritt, seinen Ursprung in der pathologischen Veranderung des Sacculus oder Utriculus hat

Um den Einfluss des Otolithensystems auf die Latenzzeit zu untersuchen haben wir zuerst die Bulla des Kaninchens von unten aufgeschnitten, den Sacculus ausgebrochen und zerstort Wie aus der Abb 7 ersichtlich, war bei der Untersuchung nach der Operation eine "DP" nach der operierten Seite zu beobachten Dabei ist die Latenzzeit auf der Seite, wo die Nystagmusdauer verlangert ist, verkurzt und auf der Seite, wo die Nystagmusdauer verkurzt ist, verlangert Namlich, die Latenzzeit verkurzt sich auf der Seite der "DP" Es weist darauf hin,

dass die Latenzzeit in einer engen Beziehung mit der Nystagmusdauer verlauft

Anschliessend daran haben wir die Grenzmembran mit Salpetersaure geatzt und den Utriculus auf einer Seite zerstort In diesem Fall verlangerte sich die Nystagmusdauer nach der gesunden Seite D H es entsteht dabei eine " DP " auf der umgekehrten Seite des Falles, bei dem der Sacculus zerstort wurde Jedoch war die Verkurzung der Latenzzeit auf der Seite der " DP " zu beobachten Es existiert dieselbe Beziehung zu dem Fall, bei dem der Sacculus zerstort wurde

Aus den obigen Untersuchungen wurde festgestellt, dass sich die Latenzzeit verlangert, wenn eine periphere CP entsteht, und dass sich die Latenzzeit auf der Nystagmusdauer verlangerten Seite verkurzt, wenn

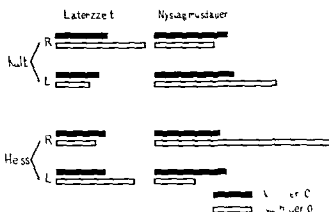


Abb 7 Veranderung der Latenzzeit und der Nystagmusdauer nach der Zerstorung des Utriculus bzw Sacculus

eine periphere " DP " durch Zerstorung des Sacculus bzw Utriculus in Erscheinung tritt Mit anderen Worten, die Latenzzeit verkürzt sich auf der Seite wo der Nystagmus leichter in Erscheinung tritt

C) *Fragen des Zentrums*

Seit langer Zeit nimmt man an dass das ubergeordnete Zentrum auf den Nystagmusreflexbogen hemmend einwirkt IVY (1919) berichtet, dass das Grosshirn den Vestibularisreflex hemmt Groebbels (1929) berichtet dass der Vermis cerebelli den Vestibularisreflex hemmt Auch Pollock u Davis (1927), Bauer u Leidler (1911) berichteten, dass das Kleinhirn den Vestibularisreflex hemmt und sich der Vestibularisreflex verstarkt wenn das Kleinhirn geschadigt wurde Crammer (1931) berichtet dass zwischen der Kleinhirnrinde, dem Kleinhirnkern und Vestibulariskern eine Gegeneinanderhemmung doppelt einwirkt Auch Mygind (1931) und Spiegel (1941—42) berichteten über Hemmwirkung des Kleinhirns

Man erklart, wenn das ubergeordnete Zentrum auf einer Seite geschadigt wird, wird die Hemmung des Vestibularisreflex auf derselben Seite beseitigt, und so tritt der Nystagmus nach derselben Seite leicht in Erscheinung, d h die sogenannte "directional preponderance" Über die zentrale Hemmwirkung haben schon unsere Mitarbeiter Takeyama u Abe (1960) durch die Untersuchungen uber die Gehirnzerstorung des Kaninchens ausfuhrlich berichtet Jedoch behandeln alle obigen Untersuchungen die Nystagmusdauer und keineswegs die Latenzzeit

Vorausgesetzt, dass das Zentrum im Vestibularissystem eine Hemmung des Nystagmus bewirkt, kann man annehmen, dass das Zentrum irgend eine Hemmschwelle auf die Latenzzeit bis zum Nystagmuseintreten durch den Labyrinthreiz hat Weiter kann man daraus schliessen, dass, wenn das Zentrum zerstort wird, es zu irgend einer Veranderung der Schwelle kommt und dass diese Veranderung selbstverstandlich die Latenzzeit beeinflusst

Deswegen haben wir das Zentrum — Lobus temporalis cerebri auf einer Seite — zerstort und beobachtet, wie sich die Latenzzeit verandert, um den zentralen Einfluss in den Fallen zu untersuchen, wo das periphere Labyrinth intakt ist

An Kaninchen wurde eine Ohrmuschel und ein ausserer Gehorgang ausgeschaltet, die knocherne Wand des Labyrinths blossgestellt und die Temperatur dieser Teile mit dem Thermister genau bestimmt Danach wurde Wasser eingespult und die Latenzzeit bestimmt Anschliessend daran wurde der Kopf aufgeschnitten, damit der Lobus temporalis des Grosshirns auf einer Seite mit dem Aspirator weggenommen werden konnte Dann haben wir Wasser unter derselben Bedingungen wieder eingespult und untersucht, wie die Latenzzeit verlauft

Abb 8 stellt die Ergebnisse dar Bei der Untersuchung, die eine Stunde nach der Zerstorung durchgefuhrt wurde, wurde beobachtet, dass, "DP" nach der gesunden Seite entstand Dabei verkürzte sich die Latenzzeit zur Richtung von "DP", namlich auf der Seite, wo sich die Nystagmusdauer verlangerte Dagegen auf der Gegenseite, wo sich die Nystagmusdauer verkurzte, war eine Verlangerung der Latenzzeit zu beobachten Dasselbe Kaninchen wurde nach 3 Stunden unter denselben Bedingungen getestet Die "DP" richtete sich diesmal nach der operierten Seite Auch bei diesem Fall war eine Verkurzung der Latenzzeit auf der Seite, wo Verlangerung der Nystagmusdauer entstand, d h zur Richtung der "DP" zu beobachten Dabei verlangerte sich die Latenzzeit auf der Gegenseite

In Bezug auf die Latenzzeit stimmen diese Ergebnisse ganz mit obigen peripheren "DP" namlich mit "DP" nach der Zerstorung des Sacculus bzw Utriculus uberein Kurz gesagt, die Latenzzeit wird auch durch die Zerstorung des Zentrums bedeutend beeinflusst und es existiert hier dieselbe Beziehung wie bei der Nystagmusdauer

Also kann man die Hemmwirkung des Zentrums auf den primären Nystagmusreflexbogen nicht nur in den Veränderungen der Nystagmusdauer, sondern auch in den Veränderungen der Latenzzeit sehen.

Das Zentrum hemmt zwar den Nystagmusreflexbogen, jedoch findet sich hier eine bestimmte Schwelle. Wenn das Zentrum irgend eine Veränderung, z. B. Zerstörung des Lobus temporalis cerebri auf einer Seite, die das Gleichgewicht zwischen beiden Vestibularissystemen stört, erfährt, tritt neben offenbarer "DP" in der Nystagmusdauer auch eine Gegenbeziehung in der Latenzzeit in Erscheinung. Dabei gibt es immer eine bestimmte Beziehung zwischen der Nystagmusdauer und der Latenzzeit zu beobachten, d. h. eine Verkürzung der Latenzzeit bei der Verlängerung der Nystagmusdauer und eine Verlängerung der Latenzzeit bei der Verkürzung der Nystagmusdauer.

Aus diesen Tatsachen ist schon klar, dass es möglich ist, durch genaue Bestimmung der Latenzzeit die "DP" oder die "CP" zu diagnostizieren.

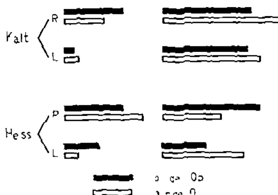


Abb. 8. Verlauf der Latenzzeit und der Nystagmusdauer nach der Zerstörung des Lobus temporalis cerebri auf einer Seite.

SCHLUSSFOLGERUNGEN

Am Kaninchen wurde die Entstehung der Latenzzeit des kalorischen Nystagmus experimentell untersucht. Dazu wurde das Vestibularissystem in a) Wärmeleitungssystem, b) Labyrinth und c) Zentrum eingeteilt und an jedem Teil erforscht. Die Ergebnisse:

a) Über das Wärmeleitungssystem.

Experimente an den äusseren Gehörgang bzw. Trommelfell ausgeschalteten Kaninchen und Erwärmungs- bzw. Abkühlungsteste des äusseren Gehörgangs wurden durchgeführt und festgestellt, dass das Wärmeleitungssystem bei Entstehung der Latenzzeit eine grosse Rolle spielt.

Weiter wurde festgestellt, dass das Warmeleitungssystem eine grosse individuelle Differenz besitzt und es daher keine Bedeutung hat, die Latenzzeit aller Individuen zu vergleichen Um zu einer bestimmten Latenzzeit zu gelangen, ist es notig, die Temperatur des Labyrinths konstant zu halten

b) Über das Labyrinth·

Wenn ein CP-Zustand durch Einspulung von Alkohol Kokain-Gemisch oder Fensterbildung des Labyrinths geschaffen wurde, war eine Verlangerung der Latenzzeit zu beobachten Wenn eine periphere "DP" durch Zerstorung des Sacculus bzw. Utriculus geschaffen wurde, verkurzte sich die Latenzzeit auf der Seite der "DP" (die Nystagmusdauer verlangerte Seite)

c) Über das Zentrum

Wenn eine zentrale "DP" durch Zerstorung des Lobus temporalis cerebri auf einer Seite geschaffen wurde, eine Verkurzung der Latenzzeit auf der Seite der "DP" (die Nystagmusdauer verlangerte Seite) beobachtet

Wie oben geschildert wurde, haben wir die Entstehung der Latenzzeit des kalorischen Nystagmus ausführlich untersucht, wie alle Teile des Vestibularissystem sich dabei beteiligen, und einige Befunde berichtet Es ist danach klar geworden, dass auch die Latenzzeit, auf die bisher kein grosser Wert gelegt wurde, diagnostisch wichtig ist, wenn sie mit einer genauen Bestimmungsmethode sorgfaltig bestimmt werden kann Besonders die Tatsache, dass Nystagmusdauer und Latenzzeit in einer bestimmten Korrelation verlaufen, scheint manche interessante Frage zu enthalten

SUMMARY

By means of demolishing various parts of vestibular system, the authors have studied experimentally on the ocurrence of latency time of the caloric nystamus

These investigations drew the following conclusions

1) The system of temperature transmission (external auditory meatus, drum membrane, temporal bone) plays an important part
2) The latency time is closely connected with the nystagmus duration
3) The true latency time has the important diagnostic value

LITERATUR

ARSLAN M 1955 On the renewing of the methodology for the stimulation of the vestibular apparatus *Acta oto-laryng Suppl 122*

INO H 1960 Clinical and experimental observations on caloric test. *Asian Medical Journal* 3 23

JONGKEES, L B W, 1949 Which is the preferable method of performing the caloric test? *Arch oto laryng 49*, 594
MITTELMAIER R, 1954 Über die Amplitude des experimentell ausgelosten Nystagmus *Pract oto rhino-laryng 16*, 108
OKAMOTO, K, 1959 Studies on caloric test for the purpose of the diagnosis of vestibular lesions *Jap Jour Otol Tokyo, 62*, 70
ONO, Y, 1960 Investigations on the caloric test *Jap Jour Otol Tokyo, 63*, 739
OWADA, K et al, 1960 The influence of the utricle on nystagmus *Acta Oto-laryng 52*, 486
SCHMALTZ, G, 1932 The physical phenomena occurring in the semicircular canals during rotatory and thermic stimulation *Proc Roy Soc Med 25* Sec Otol 359
STAHLE, J, 1958 Electronystagmography in the caloric and rotatory tests *Acta oto laryng Suppl 137*
WODACK, E, 1952 Diagnostic value of the latency period after caloric stimulation of vestibular apparatus *Arch Otolaryng 55*, 3

Meguroku Tokyo, Japan

THE EFFECT OF EYE CLOSURE UPON NYSTAGMUS

TOSHI NAITO TOSHIFUMI TATSUMI TORU MATSUNAGA
and TAKASHI MATSUNAGA
Osaka Japan

From the Department of Otorhinolaryngology (Head Prof Dr T Hasegawa) Osaka University School of Medicine Osaka

Influence of closure of eye and mental arithmetic on nystagmus was studied Influences on nystagmus during horizontal pendular rotation (Amp 45° Period 5) In cases of the normal and central lesion Nystagmus is inhibited by closure of eye and this inhibition is removed by mental arithmetic

Closure of eye makes caloric nystagmus larger in the normal persons and the patients with the peripheral lesion and smaller in the patients with the central lesion

Spontaneous or positional nystagmus is inhibited by closure of eye in cases with central lesion such as brain tumor but not in cases with the peripheral lesion such as Menieres disease

Nystagmus inhibited by closure of eye becomes apparent by mental arithmetic

The data may be useful to identify the lesion whether it is central or peripheral

It is well known that nystagmus has been divided into two kinds optic and vestibular according to the organ from which its provoking impulse primarily originates The fact that both kinds of nystagmus were essential for optic perception was also shown (V Holst 1956)

Henriksson in 1957 found that the velocity of slow component of nystagmus was controled by vestibular stimulation and that simultaneous participation of impulses from both optic and vestibular organ was necessary to maintain the fixation of vision This fixation of vision makes up the slow component of nystagmus when one is subjected to a rotation with eyes open In the absence of impulses from both optic and vestibular organ fixation of vision did not occur This Henriksson s observation gave a definite assurance to the idea that cooperative working of optic and vestibular organ was essential for optic perception In our country Fukuda is of opinion that optic stimulation is primarily responsible for the occurence of nystagmus while vestibular stimulation is of secondary significance

Optic influence upon vestibular nystagmus has been discussed in detail from various aspects Examining caloric nystagmus with ENG

in the following 4 conditions, on eye-opening in a bright room, on wearing Frenzel's spectacles, on eye opening in a dark room and on eye-closure, Mahony (1957) noted an increased amplitude and a decreased frequency of nystagmus on closure of eyes There is no reported practical use of these observations

As it was considered to be an interesting way of approach to study the effect of optic stimulation and mental work upon nystagmus, an electroencephalographical study was made to give a better understanding to the difference of nystagmus in quality as well as in quantity in both states of eye opening and eye closure In the latter state, optic influence was to be actively eliminated As the method of vestibular stimulation was employed a horizontal pendular rotation (amplitude 45 degrees, period 5 seconds), because it was considered to be the most "physiological" according to Jongkee's theoretical and Bekesy's experimental study For the method of caloric stimulation, was chosen Hallpike and Fitzgerald's method which offered relatively long duration of nystagmus It was mainly for the purpose of excluding influence from CNS that those methods of relatively weak stimulation were chosen

The result and the clinical application will be mentioned in the following paragraphs

SUBJECT AND METHOD

30 persons were selected out of those who visited the Out Patient Clinic of Osaka University Hospital with chief complaint of vertigo, while control group consisted of 10 persons chosen from doctors and nurses working at the Clinic

Method ENG was used to record the nystagmus during a rotatory movement of the subject sitting on an semi electromotive pendular rotation chair with amplitude of 45 degrees and period of 5 seconds, in both states of eye opening in a bright room and eye closure At the same time brain wave was recorded by parietooccipital lead of EEG In some cases effect of mental arithmetic on both nystagmus and brain wave was examined Caloric nystagmus was, together with brain wave, recorded in both repeated alternate states of eye-opening in a bright room and of eye closure following an irrigation of an external auditory canal with 50 cc of 30 degree water Further spontaneous and postural mystagmus, if present were recorded to examine the difference in both states of eye-opening and eye-closure

RESULTS

(1) Pendular rotation As shown in Fig 1, normal persons sitting, with their eye opening, on the above mentioned pendular rotation chair showed average 6 to 7 nystagmus per one swing to the opposite directions

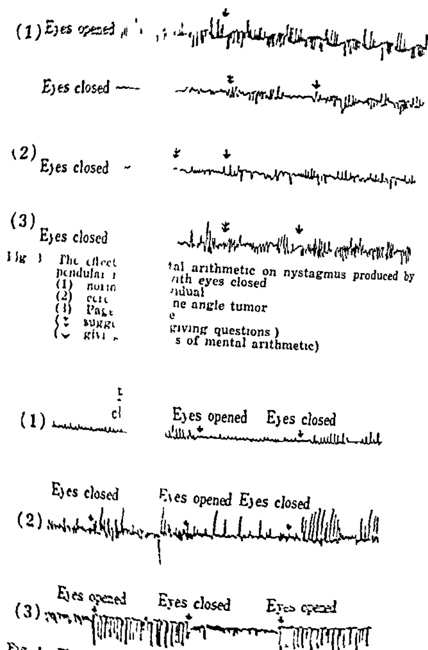


Fig 3 The effect ... tal arithmetic on nystagmus produced by pendular r... ith eyes closed
(1) norm... idual
(2) cere... ne angle tumor
(3) Page... e
{ sugge... giving questions)
(give... s of mental arithmetic)

Fig 4. The effect of closure of eyes on caloric nystagmus irrigation with 30°c, 50 cc water.
(1) Normal individual
(2) Meniere's disease
(3) Cerebellopontine angle tumor

... as well as nystagmus observed during the rotation were found ... in two different ways by closure of eyes. Study of other ... ms and symptoms revealed that one group was frequent[illegible] ... central disorders and the other with peripheral disorders ... former "central type" and the latter "peripheral type". Details are summarized in Table 1.

in the following 4 conditions, on eye opening in a bright room, on wearing Frenzel's spectacles, on eye-opening in a dark room and on eye closure, Mahony (1957) noted an increased amplitude and a decreased frequency of nystagmus on closure of eyes There is no reported practical use of these observations

As it was considered to be an interesting way of approach to study the effect of optic stimulation and mental work upon nystagmus, an electroencephalographical study was made to give a better understanding to the difference of nystagmus in quality as well as in quantity in both states of eye opening and eye closure In the latter state, optic influence was to be actively eliminated As the method of vestibular stimulation was employed a horizontal pendular rotation (amplitude 45 degrees, period 5 seconds), because it was considered to be the most "physiological" according to Jongkee's theoretical and Bekesy's experimental study For the method of caloric stimulation, was chosen Hallpike and Fitzgerald's method which offered relatively long duration of nystagmus It was mainly for the purpose of excluding influence from CNS that those methods of relatively weak stimulation were chosen

The result and the clinical application will be mentioned in the following paragraphs

SUBJECT AND METHOD

30 persons were selected out of those who visited the Out-Patient Clinic of Osaka University Hospital with chief complaint of vertigo, while control group consisted of 10 persons chosen from doctors and nurses working at the Clinic

Method ENG was used to record the nystagmus during a rotatory movement of the subject sitting on an semi electromotive pendular rotation chair with amplitude of 45 degrees and period of 5 seconds, in both states of eye-opening in a bright room and eye closure At the same time, brain wave was recorded by parietooccipital lead of EEG In some cases, effect of mental arithmetic on both nystagmus and brain wave was examined Caloric nystagmus was, together with brain wave, recorded in both repeated alternate states of eye-opening in a bright room and of eye-closure, following an irrigation of an external auditory canal with 50 cc of 30 degree water Further, spontaneous and postural mystagmus, if present, were recorded to examine the difference in both states of eye-opening and eye closure

RESULTS

(1) Pendular rotation As shown in Fig 1, normal persons sitting, with their eye opening, on the above mentioned pendular rotation chair showed average 6 to 7 nystagmus per one swing to the opposite directions

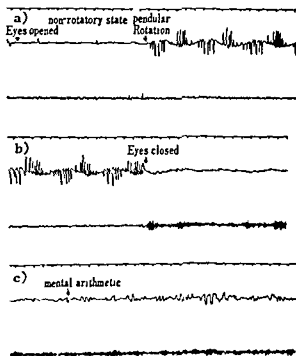


Fig 1 Simultaneously recorded Eye Speed Curve of Nystagmus and Parietooccipital Lead of EEG in a normal person; a, b and c traced left to right. The upper curve of a, b and c is in respective Eye Speed Curve of Nystagmus, time constant of 0 03 sec, and the lower is EEG, time constant of 0 3 sec, the both, paper speed of 1 5 cm per sec.

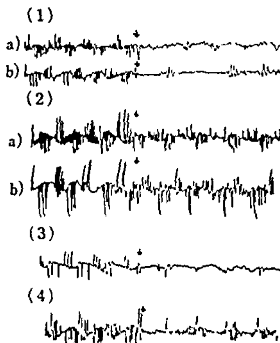


Fig 2. The effect of closure of eyes on nystagmus produced by pendular rotation, all eye speed curves, time constant of 0 03 sec, paper speed of 0 5 cm per sec (⌄ closure of eyes)
(1) 2 normal examples
(2) Menière's disease (2 cases)
(3) Cerebello-pontine angle tumor
(4) Paget's disease

ernatively Simultaneously recorded parietooccipital lead of EEG did t make any remarkable change to the brain wave observed in the non tatory state of eye opening EEG in this rotatory state of eye opening is characterized by many low and fast waves Closure of eyes resulted the disappearance of nystagmus and the appearance of an ocular move ent synchronized to the pendular rotation, while closure of eyes resulted the appearance of high 10 cps brain waves When mental arithmetic as ordered for the purpose of adding mental work to this state of eye osure, nystagmus showed a reappearance and an alpha blocking occured EEG at the beginning of mental arithmetic

When the same procedure was done to those subjects with chief com aint of vertigo, nystagmus showed two kinds of changes while EEG dings were essentially same as those in normal individuals Namely, shown in Fig 2, on closure of eyes on rotation chair, nystagums almost mpletely disappeared in some individuals just as did in control group Fig 2, 3, 4), while it remained unchanged in some cases (Fig 2 a and b)

In the former, mental arithmetic exerted an influence upon nystagmus it not in the latter Fig 3 is an example

(2) Caloric stimulation Following an irrigation of an external iditory canal with 50 cc of 30 degree water in 10 seconds in eye opened, ipine position of the subject with his head anteflexed by 30 degrees pro- oked caloric nystagmus was recorded on alternate states of eye opening id eye-closure As shown in Fig 4 normal persons usually showed ister nystagmus on closure of eyes but some of them showed suppression n the other hand, in those subjects complaining of vertigo there were ome cases of decreased nystagmus on closure of eyes as well as those ises of increased nystagmus just as seen among normal persons Simul ineously recorded EFG exhibited an appearance of alpha wave on closure f eyes and alpha blocking on opening of eyes As shown in Fig 5, mental rithmetic as a mental work did not exert any definite influence upon ystagmus regardless of the state of eye opening or eye-closure Com ination of eye closure and mental arithmetic produced, in some cases, irger nystagmus than eve opening did though mental arithmetic alone iiled to cause any change to nystagmus (Fig 5, 3)

(3) Spontaneous and Postural nystagmus About the subjects with hief complaint of vertigo spontaneous and postural nystagmus were re- orded with ENG in both states of eye opening and eye closure in sitting osition Result was either an increase or a decrease of nystagmus on losure of eyes Fig 6 is an example

When mental arithmetic as a mental work was added to the state of ye-closure some subjects showed increased spontaneous and postural ystagmus (Fig 7) Table 1 is the summary of the study done from the bove-mentioned three different aspects

30 subjects who had a complaint of vertigo were devided into two groups according to the fact that their spontaneous postural and caloric

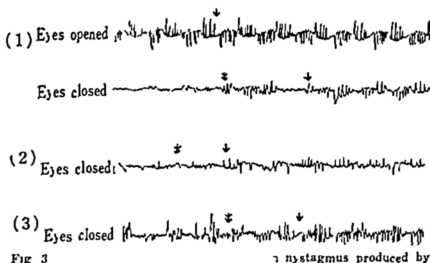


Fig 3 ı nystagmus produced by

(3) Paget s disease
(⇟ suggestion of giving questions)
(⌄ giving questions of mental arithmetic)

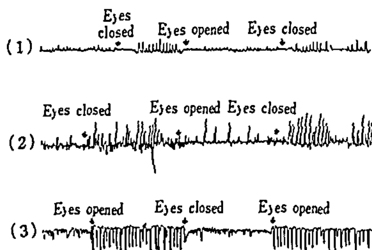


Fig 4 The effect of closure of eyes on caloric nystagmus, irri gation with 30°c, 50 cc water
(1) normal individual
(2) Meniere s disease
(3) Cerebello pontine angle tumor

nystagmus as well as nystagmus observed during the rotation were found to be influenced in two different ways by closure of eyes Study of other neurological signs and symptoms revealed that one group was frequently associated with centr il disorders and the other with peripheral disorders Auther namcd the former ' central type " and the later, " peripheral type " *for convenience Details are summarized in Table 2*

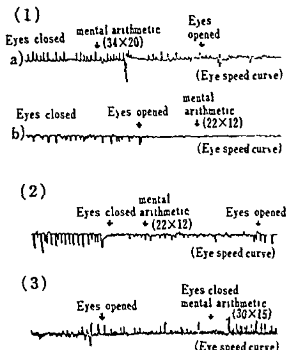


Fig. 5. The effect of mental arithmetic on caloric

gation of the right ear.

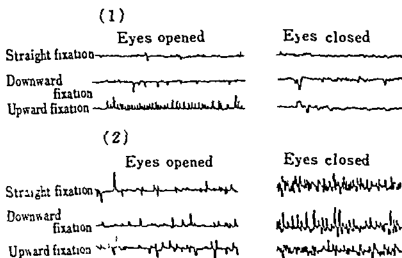


Fig 6 The effect of closure of eyes on positional nystagmus.
(1) Cerebello-pontine angle tumor, left lateral flexion of head
(2) Meniere's disease, left lateral flexion of head.

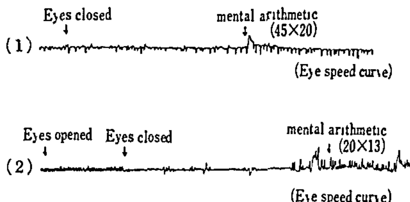


Fig 7 The effect of mental arithmetic on positional nystagmus in patient with Paget's disease
(1) left lateral flexion of head
(2) right lateral flexion of head

Table 1. *Change of Nystagmus with closure of eyes*

Method of stimulation	Pendular rotation		Cold stimulus	Postural change of head
Condition	Eyes closed	Calculation performed with eyes closed	Eyes closed	Eyes closed
Kinds of patient examined				
Normal type	Nystagmus disappeared	Nystagmus reappeared	Nystagmus increased	—
Central type	Nystagmus disappeared	Nystagmus reappeared	Nystagmus decreased	Nystagmus disappeared
Peripheral type	Nystagmus not disappeared	Nystagmus unchanged	Nystagmus increased	Nystagmus increased

SUMMARY AND COMMENT

It has been quite well established that optic stimulation exerts considerable influence upon vestibular responses Rossberg found that spontaneous nystagmus got easier in its appearance in the following ascending order, on eye opening in a bright room, eve opening in a dark room and eye-closure We also found larger spontaneous nystagmus in a dark room than in a bright room in rabbits whose semicircular canals had been destroyed unilaterally

Hallpike, Aschan (1958), Mahoney (1957) and Minakawa (1959) had found that closure of eyes caused increased intensity and prolonged duration of caloric nystagmus in human subjects This fact is indicative of that removal of optic stimulation has an activating effect on the intensity

of nystagmus On the other hand, as our results show, the closure of eyes is considered to have an inhibitory effect on nystagmus which is observed during pendular rotatory movement

Using EMG, Van Eyck (1960) found that passive removal of optic stimulation had an activating effect upon postrotatory nystagmus Above-mentioned can be summarized as that sponteneous, caloric and postrotatory nystagmus can be activated and the nystagmus which is observed during rotatory movement can be inhibited by the removal of optic stimulation In other words, optic organ which helps recognizing one's posture in the space, has either activating or inhibitory effect on vestibular nystagmus In the term of removal of optic stimulation are included eye opening in a dark room, deprived fixation of vision with Frenzel's spectacles and eye-closure

In the preceding paragraphs, author reported an observation that 10 normal persons, when exposed to the pendular rotation, showed decreasing tendency of nystagmus in its frequency and intensity in the state of eye opening in a bright room, on wearing Frenzel's spectacles, eye-opening in a dark room and in the state of eye-closure in the ascending order Details are shown in Table 3

It is understood from this Table that frequency of nystagmus and its velocity in slow component showed relatively minor differences in the state of eye-opening in a bright room, on wearing Frenzel's spectacles and in the state of eye opening in a dark room, but that they decreased to such an extent on eye-closure that their ENG recording became impossible

Use of Frenzel's spectacles and the state of eye-opening in a dark room can be thought to be a "passive" removal of optic stimulation while eye-closure is its "active" removal Therefore it seems to be reasonable to assume that there exists an essential difference between the two phenomena of eye opening and eye-closure

To investigate the fundamental cause which might be present between the difference of "passive" and "active" removal of optic stimulation, simultaneous recording of ENG and parietooccipital lead of EEG was performed about subjects exposed to the pendular rotation on a semi electromotive rotation chair This experiment revealed that appearance of large brain waves with frequency of about 10 cps (alpha wave) and disappearance of nystagmus occured on eye-closure while brain wave was small in amplitude and moderate in frequency (alpha blocking) and nystagmus was of marked intensity when eyes were opened (Fig 1)

When unilateral semicircular canals were stimulated with 50 cc of 30 degree water eye closure and eye-opening, respectively, exerted their influence upon brain wave in the same fashion as they did in pendular rotation test in contrast to their reverse influence upon nystagmus

From these facts their seem to exist a certain relationship between nystagmus, a subcortical reaction and brain wave, a cortical bioelectric

Table 2 *Change of Nystagmus with closure of eyes*
Cases of central type (No 1)

Case examined	Diagnosis	Chief Complaint	Spontaneous Nystagmus	Postural Nystagmus	Cold Stimulation	Pendular Rotation Test
1	Perceptive deafness	Impaired hearing Tinnitus			Nystagmus decreased	Nystagmus disappeared
2	Brain Tumor	Vertigo Tinnitus			Nystagmus decreased	Nystagmus disappeared
3	Posttraumatic perceptive deafness	Tinnitus			Nystagmus decreased	Nystagmus disappeared
4	Cerebral hemorrhage (Hypertension)	Vertigo Tinnitus			Nystagmus decreased	Nystagmus disappeared
5	Paget's *disease*	Impaired hearing Disturbance *of gait* Dysarticulation	Nystagmus *decreased*	Nystagmus *decreased*	Nystagmus *decreased*	Nystagmus *disappeared*
6	Cerebello-pontine angle tumor	Vertigo Tinnitus perceptive deafness paraesthesia of face	Nystagmus decreased	Nystagmus decreased	Nystagmus decreased	Nystagmus disappeared
7	Tubular sclerosis	Headache Tinnitus Visual disturbance	Nystagmus decreased		Nystagmus decreased	Nystagmus disappeared
8	Cerebro-spinal Meningitis	Disturbance of equilibrium	Nystagmus decreased	Nystagmus decreased	Nystagmus decreased	Nystagmus disappeared
9	Vertigo	Vertigo Impaired hearing			Nystagmus decreased	Nystagmus disappeared
10	Brain tumor	Vertigo Impaired hearing Disturbance of equilibrium	Nystagmus decreased	Nystagmus decreased	Nystagmus decreased	Nystagmus disappeared
11	Vertigo	Tinnitus Vertigo			Nystagmus decreased	Nystagmus disappeared

Cases of peripheral type (No 2)

Case examined	Diagnosis	Chief Complaint	Spontaneous Nystagmus	Postural Nystagmus	Cold Stimulation	Pendular Rotation Test
1	Meniere's disease	Vertigo Tinnitus Impaired hearing	Nystagmus increased	Nystagmus increased	Nystagmus increased	Nystagmus not disappeared
2	Meniere's disease	Vertigo Tinnitus. Impaired hearing			Nystagmus increased	Nystagmus not disappeared
3	Vertigo	Vertigo	Nystagmus increased	Nystagmus unchanged	Nystagmus increased	Nystagmus not disappeared
4	Meniere's disease	Vertigo Tinnitus Impaired hearing Headache			Nystagmus increased	Nystagmus not disappeared
5	Meniere's disease	Vertigo Tinnitus Impaired hearing			Nystagmus increased	Nystagmus not disappeared
6	Meniere's disease	Vertigo Tinnitus Impaired hearing	Nystagmus increased	Nystagmus increased	Nystagmus increased	Nystagmus not disappeared
7	Tinnitus of neural origin	Vertigo Impaired hearing			Nystagmus increased	Nystagmus not disappeared
8	Meniere's disease	Vertigo Tinnitus Impaired hearing			Nystagmus increased	Nystagmus not disappeared
9	Meniere's disease	Vertigo Tinnitus. Hardness of hearing			Nystagmus increased	Nystagmus not disappeared
10	Vertigo Tubal stenosis	Vertigo Tinnitus. Hardness of hearing			Nystagmus increased	Nystagmus not disappeared
11	Vertigo	Vertigo Nausea			Nystagmus increased	Nystagmus not disappeared
12	Meniere's disease	Tinnitus. Vertigo Impaired hearing Heavy head			Nystagmus increased	Nystagmus not disappeared
13	Vertigo	Vertigo	Nystagmus increased	Nystagmus increased	Nystagmus increased	Nystagmus not disappeared
14	Bulbar palsy	Dysarticulation Dysphagia			Nystagmus increased	Nystagmus not disappeared

Case examined	Diagnosis	Chief Complaint	Spontaneous Nystagmus	Postural Nystagmus	Cold Stimulation	Pendular Rotation Test
15	Perceptive deafness	Impaired hearing			Nystagmus increased	Nystagmus not disappeared
16	Palsy of trigeminal Nerve (L) Palsy of abducent Nerve (L)	Anaesthesis of left half of face			Nystagmus increased	Nystagmus not disappeared

Cases of unclassified type (No 3)

Case examined	Diagnosis	Chief Complaint	Spontaneous Nystagmus	Postural Nystagmus	Cold Stimulation	Pendular Rotation Test
1	Perceptive deafness	Heavy head Vertigo Impaired hearing			Nystagmus increased	Nystagmus disappeared
2	Tinnitus of neural origin	Vertigo Tinnitus	Nystagmus decreased	Nystagmus decreased	Nystagmus increased	Nystagmus disappeared
3	Undetermined	Disturbance of gait			Nystagmus decreased	Nystagmus not disappeared

Table 3. *Change of Nystagmus with pendular rotation*

Nystagmus	Condition examined: Eyes opened in bright room	with Frenzel's glasses in bright room	Eyes opened in dark room	Eyes closed
Beat number	5.7	6.1	4.8	0
Relative eye-speed of slow phase	4.6	3.5	3.5	0

reaction but no direct correlation Adrian, Spiegel, Ouchi (1951) and Motokawa (1947) found alpha blocking on EEG occuring simultaneously with Barany's postrotatory nystagmus In cur experiment, however, pendular rotation did not cause any electroencephalographic change in both states of eye opening and eye-closure with an only exception of temporary appearance of a weak alpha blocking in a state of eye closure immediately after the beginning of pendular rotatory movement That alpha blocking disappeared again after several pendular rotations

Those different results in the above-mentioned two observations may be due to the difference in intensity of stimulation and different time of observations done (during or after the rotation) Concerning the relationship between nystagmus and brain wave it is to be emphasized again here that eye closure exerts its influence upon simultaneously provoked brain wave and rotatory or caloric nystagmus in a certain difinite way of regularity Namely, regular combinations of alpha blocking with increased nystagmus and alpha wave with decreased nystagmus were demonstrated in a series of our previously mentioned experiments, pendular rotation test on eye opening, on eye-closure and on mental work in addition of eye closure This rule holds true only in pendular rotation test but a reverse relationship could be demonstrated between caloric nystagmus and brain wave Such a definit effect of mental work could not be demonstrated in caloric stimulation

Consequently, it is easily understood that mental arithmetic which caused arousal reaction in brain wave has an important effect on nystagmus occuring during pendular rotation Nystagmus is as well known, a rhythmic ocular movement, consisting of slow and fast component Sub cortical vestibulo-ocular system has been claimed as a provoking mechanism of fast component of nystagmus

In our experiment, an ocular movement which was opposite to the pendular rotation in its direction was still found on eye clesure, though the nystagmus observed during pendular rotation was markedly decreased on eye-closure If this ocular movement is regarded as the primary vestibulo-ocular reflex, it is quite natural that mental arithmetic as a mental work should cause arousal reaction in brain wave and at the same time participate in provoking the fast component of nystagmus which is considered to be central in origin

In support of the above-mentioned idea, there are two reports, Magoun found that a portion of reticular formation was responsible for arousal reaction in brain wave and Lorente de No (1933) reported disappearance of fast component of nystagmus on destruction of reticular formation When above mentioned are put together it becomes quite clear that besides the absence of optic stimulation central mechanism which can be influenced by mental work is involved in the effect of eye-closure upon nystagmus Then, if eye closure was taken as a standard state, eye opening was found

15	Perceptive deafness	Impaired hearing			Nystagmus increased	Nystagmus not disappeared
16	Palsy of trigeminal Nerve (L) Palsy of abducent Nerve (L)	Anaesthesis of left half of face			Nystagmus increased	Nystagmus not disappeared

Cases of unclassified type (No 3)

Case examined	Diagnosis	Chief Complaint	Spontaneous Nystagmus	Postural Nystagmus	Cold Stimulation	Pendular Rotation Test
1	Perceptive deafness	Heavy head Vertigo Impaired hearing			Nystagmus increased	Nystagmus disappeared
2	Tinnitus of neural origin	Vertigo Tinnitus	Nystagmus decreased	Nystagmus decreased	Nystagmus increased	Nystagmus disappeared
3	Undetermined	Disturbance of gait			Nystagmus decreased	Nystagmus not disappeared

Table 3 *Change of Nystagmus with pendular rotation*

Nystagmus	Condition examined: Eyes opened in bright room	with Frenzel's glasses in bright room	Eyes opened in dark room	Eyes closed
Beat number	5.7	6.1	4.8	0
Relative eye-speed of slow phase	4.6	3.5	3.5	0

Augenschliessen vergrossert kalorischen Nystagmus mehr in normalen Fallen und derartigen mit peripheraler Schadigung, und ist kleiner in zentraler

Spontane- und Lage-Nystagmus wird durch Augenschliessen bei zentraler Schadigung (ZB Hirn Tumoren) gehemmt, aber nicht in peripheraler (ZB Menièresche Krankheiten)

Gehemmter Nystagmus durch Augenschliessen erscheint bei Kopfrechnung ziemlich deutlich zu sein

Diese Data gelten, um dem Herd, zentral oder peripheral, zu bestimmen

REFERENCE

ASCHAN, G, 1958 Different type of alcohol nystagmus *Acta oto laryng Suppl 140*

BÉKÉSY, G V, 1955 Subjective cupulometry—Threshold, adaptation, and sensation intensity of the vestibular organ for rotations in the horizontal plane *Arch oto laryng 61*, 16

van EGMOND, A A J, GROEN J J and L B W JONGKEES 1952 *The Function of the vestibular organ*, S Karger Basel Schweiz

van EYCK M 1960 Effects of illumination of the eye on the vestibular responses in the pigeon, *Acta Oto-laryng, 51* 421

FUKUDA, T, 1957 *Stato kinetic reflexes in equilibrium and movement* Igaku Shoin Ltd Tokyo

GOTTESBERGE, A, Meyer zum, 1957 Nystagmus und Richtungskonstanz, *Arch Ohr usw Heilk, 172*, 78

HALLPIKE, C S, FITZGERALD, G, and J E CAWTHORNE 1942 Studies in human vestibular function *Brain, 65*, 115

HALLPIKE, C S, HARRISON M S and E SLATOR, 1951 Abnormalities of the caloric test results in certain varieties of mental disorder *Acta oto laryng 39*, 151

HENRIKSSON N G, 1955 The correlation between the speed of the eye in the slow phase of nystagmus and vestibular stimulus *Acta oto-laryng, 45*, 120

HENRIKSSON, N G, 1957 Speed of slow component and duration in caloric nystagmus *Acta oto laryng, Suppl 125*

JUNG R, 1948 Die Registrierung des postrotatorischen und optokinetischen nystagmus und die optisch vestibulare integration beim Menschen *Acta otolaryng 36* 199

LORENTE de No, R 1933 Vestibulo-ocular reflex arc *Psychiatry, 30*, 245

MAGOUN, H W, 1958 *The waking Brain* C C Thomas Pub, Springfield, U S A

MAHONEY, J L, HARLAN W L and R G BICKFORD 1957 Visual and other factors influencing caloric nystagmus in normal subjects *Arch otolaryng 66*, 46

MINAKAWA, I, 1959 Effects of visual function upon caloric nystagmus *Jap Jour Otol, 62*, 690

MOTOKAWA, K, 1947 *Brain wave* Nanjo Shoten Ltd Tokyo

OUCHI, J, 1951 Studies on the influence of the rotatory stimulation of labyrinth upon normal human brainpotentials *Jap Jour Otol 54* 187

ROSSBERG, G, 1954 Spontannystagmus und Drehschwindel *Arch. Ohr Nas u. Kehlk, 165* 417

WÜST, H, 1956 Untersuchungen des Vestibularapparates mit neuen Drehschwachreizmethoden *Arch Ohr Nas u. Kehlk 168* 414

Dojima Hamadori Fukushima ku Osaka

SPONTANEOUS NYSTAGMUS AS A SIGN OF CLINICAL SIGNIFICANCE

ICHIRO KIRIKAE, JUN ICHI SUZUKI and KOJI TOKUMASU
Tokyo, Japan

From the Department of Otorhinolaryngology, Faculty of Medicine (Head Prof. Ichiro Kirikae M D) University of Tokyo, Tokyo

In the examination of patients suffering from vertigo of loss-of-balance, stress should be put on demonstration and identification of spontaneous nystagmus The order of applied test procedures of spontaneous nystagmus is based upon experience gained in examining patients in our clinic

The applied order is as follows -

1) examination of spontaneous nystagmus with the eyes open,
2) examination of spontaneous nystagmus behind Frenzel's spectacles,
3) examination of spontaneous nystagmus by using electronystagmography,
4) optokinetic pattern test (OKP test),
5) mobilization of spontaneous nystagmus through optokinetic stimulation

The first four above procedures have their respective importance and should be performed as a matter of routine in all vertigo or loss of-balance cases The third, fourth and fifth tests are performed by using electronystagmography, the clinical significance of which cannot be over-emphasized in optokinetic pattern test, as it is termed in our clinic is helpful both for differentiation of spontaneous nystagmus and quantitative assessment of the intensity of spontaneous vestibular nystagmus The final procedure, in which optokinetic after-nystagmus is compared between that of left and right, is performed especially in cases where spontaneous nystagmus is suspected but not definitely demonstrated in the preceding four procedures

INTRODUCTION

There have been many works on the problem of spontaneous nystagmus (Frenzel 1955, Morimoto 1955) In spite of many excellent works in the fields of neurology, oto- and ophthalmo-neurology, its diagnostic importance does not yet appear to be widely appreciated This report is on how to examine spontaneous nystagmus, especially that from peripheral vestibular lesion

While no one will deny the importance of demonstration of spontaneous nystagmus including positional nystagmus (Morimoto 1955, Nylen 1931, Lindsay 1951, Dix & Hallpike 1952, Stenger 1955) in examination

of patients suffering from vertigo or loss of-balance, not all appear to put this principle into practice Frenzel (1955), among others, should be highly appreciated for his simple but valuable device for observing nystagmus Frenzel's spectacles are very useful both for detection of peripheral vestibular nystagmus and for examination of positional and positioning nystagmus

Electronystagmography, making use of corneoretinal potential, is now becoming and indispensable apparatus for vestibular examination, because of its simple and accurate recording technique and providing possible nystagmus recording in darkness or with the eyes closed The only weak point of ENG is that it cannot record rotatory type nystagmus, but advantages of ENG in general, however, much more than make up for this weakness ENG has made possible exact recording of experimental nystagmus induced by optokinetic, rotatory or caloric stimulation Thus, through electronystagmographic technique, examination utilizing nystagmic reaction has actually become as important as EKG and EEG in clinical test (Aschan, Bergstedt & Stahle 1956, Jongkees 1960)

Optokinetic nystagmus is such a remarkable phenomenon that many investigators have engaged in fundamental and clinical studies of this problem Its clinical application, however, has been limited up to the present By using both electrically controlled rotating drum and ENG, the authors want to show that there is a distinct possibility of optokinetic nystagmus being widely utilized by clinicians as one of the routine tests (Suzuki 1962)

EXAMINATIONS

In our clinic, the following steps are taken for examination of spontaneous nystagmus –

1) *Examination of spontaneous nystagmus appearing in opened eyes, i e, intent fixation*

Nystagmus is observed when the eyes are fixed on the examiner's index c 50 cm apart Both eyes are examined for ocular movements and nystagmus Demonstration of gaze nystagmus is important in diagnosis Lateral gaze nystagmus, when it appears asymmetric, has diagnostic significance

2) *Examination of spontaneous nystagmus under Frenzel's spectacles*

The first purpose of such an examination is to detect vestibular spontaneous nystagmus which is usually so small in amplitude that its observation requires great attention and care Patients are required to look in a straight line at a distant object

Positional and positioning nystagmus are also examined behind Frenzel's spectacles If positional or positioning nystagmus is suspected from conditions of which a patient complains, a detailed test in more than

ten different positions and positionings along horizontal, sagittal and diagonal planes in advocated (Sakata in press). A positional test is made with the patient in a sitting position. In cases requiring detailed examination, a positional test is made with the patient in a lying position. Tests, in a sitting position, are made in all cases. Changing of position is done as slowly as possible in a positional test, but as quickly as possible in a positioning test. When a patient complains of a particular position or positioning is important and significant for establishing diagnosis (Sakata in press).

3) *Examination of spontaneous nystagmus by using electronystagmography*

Nystagmus is recorded electronystagmographically with the eyes open, closed, and covered (Fig. 1). It is not uncommon to demonstrate by means of ENG nystagmus which cannot be confirmed by observation with the eyes open or behind Frenzel's spectacles. Adequate measures are taken to made doughtful nystagmus appear distinct in ENG recording: elevation of activity level of the brain, for example, through "calling

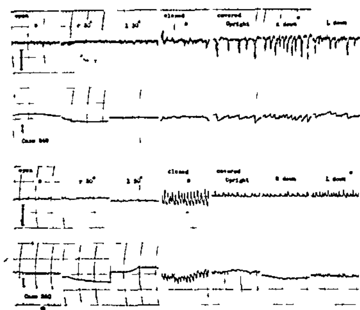


Fig 1 Electronystagmographic recording of spontaneous vestibular nystagmus in two cases; eyes open, closed and covered, respectively. Nystagmus appears most distinct, with the eyes covered, in case No 248; with the eyes closed, in case No 260
Calibration shows 100 μV
S looking straight forward
r 30° lateral gazing to the right by 30 degrees
l 30°. lateral gazing to the left by 30 degrees
Upright head upright
R down head inclined to the right
L-down· head inclined to the left
a , b curves of eye deviation
a', b' curves of eye-speed

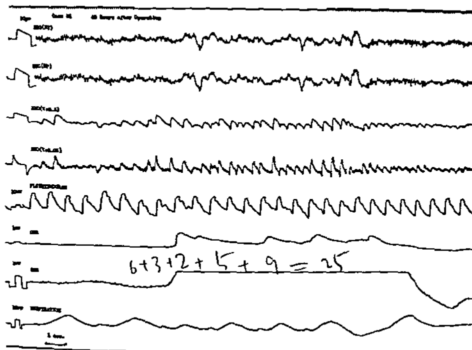


Fig 2 Polygraphic recording in a case with the eyes closed, 48 hours after left labyrinth destruction Spontaneous nystagmus appears distinctly during applied mental calculation

to the patient", "hand-clapping", "telling him to make a mental calculation", "changing the respiratory condition", etc, often affords provocation of spontaneous nystagmus (Fig 2)

4) *Optokinetic pattern test (OKP test)*

Optokinetic nystagmus is provoked by an electrically controlled rotating drum, and recorded by ENG Rotation of the drum is accelerated from zero to a certain angular velocity (100°/sec or 180°/sec. is chosen) with a constant acceleration of 4°/sec², and immediately decelerated to zero with an idential but negative acceleration This is followed by another rotation in the opposite direction Induced nystagmus is recorded by ENG on the paper fed with 0 1 cm/sec, giving characteristic patterns, as illustrated (Fig 3) It is necessary to compare patterns obtained from rotations in both directions

5) *Mobilization (activation) of spontaneous nystagmus through optokinetic stimulation*

Optokinetic nystagmus is provoked by the rotating drum The drum is rotated with a constant angular acceleration of c 1°/sec² up to a certain angular velocity where optokinetic nystagmus is most actively and constantly provoked The optimum velocity differs from case to case: 30° to 100°/sec or more When the optimum angular velocities differ between right and left rotations, the lower one is chosen as the maximum

speed to apply to the test-subject After-nystagmus is recorded by suddenly terminating optokinetic stimulus, i e, by covering or closing the eyes After-nystagmus induced by right rotatory stimulus is compared with that after the same amount of left rotatory stimulus (Fig. 4).

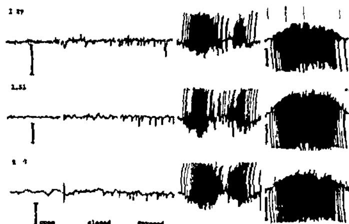


Fig 3 Electronystagmographic recording of spontaneous vestibular nystagmus and OKP in a case after acute depression of function of the right labyrinth spontaneous nystagmus directing to the light decreased day by day Optokinetic patterns are influenced by spontaneous nystagmus Asymmetry in optokinetic patterns is shown to decrease
r OKP Optokinetic pattern of optokinetic nystagmus provoked towards the right
l OKP Optokinetic pattern of optokinetic nystagmus provoked towards the left

COMMENTS

As mentioned in the "Introduction", demonstration and identification of spontaneous nystagmus has profound diagnostic importance The first and second procedures referred to this paper are actually supposed to be put into practice in vertigo clinics, and are expressively stated to show the order thereof and mention their important points Three test-procedures were added by authors to additions, last two procedures are optokinetic nystagmus-tests Optokinetic stimuli do not directly act upon the peripheral labyrinths, therefore, enabling vestibular function to be studied in the state of static counterbalance

1) Examination of spontaneous nystagmus appearing in opened eyes

Examination of ocular movements under visual fixation is important for differential diagnosis of vestibular nystagmus from central or ocular type of nystagmus, e g, nystagmus of peripheral labyrinthine origin is

direction fixed and usually so small in amplitude that its recognition with the naked eye is usually difficult, gaze nystagmus with large amplitude going to both sides does not belong to nystagmus of peripheral labyrinthine origin, neither does nystagmus of large amplitude without accompanying vertigo belong to peripheral labyrinthine origin

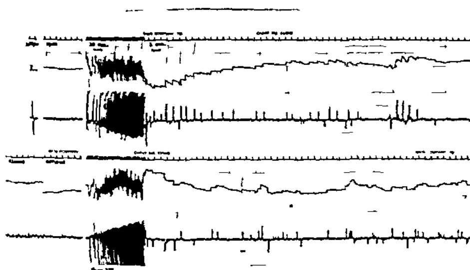


Fig 4

left (*)

2) Examination of spontaneous nystagmus under Frenzel's spectacles

Frenzel's spectacles deprives, though not completely, visual fixation and, through this, facilitates the appearance of spontaneous nystagmus hardly recognizable with the unaided eye

Closer observation of positional or positioning nystagmus concerning direction, amplitude, frequency latency, duration, and accompanying vertigo will serve, by presenting more than auxilliary data, for differential diagnosis of nystagmus and causative lesions Positional and positioning nystagmus appears to have characteristics according to the site of lesion Nystagmus appearing in cases of acoustic trauma, Meniere's disease, head injury, tumour in the posterior cranial fossa, and in so-called benign paroxysmal positional vertigo constitute characteristic groups of positional and positioning nystagmus

3) Examination of spontaneous nystagmus by using electronystagmography

Electronystagmography utilizing corneoretinal potential, according to the author's view, has brought great progress in the field of vestibular examination In the same way as science begins by accurate registration

and makes considerable advance by improvement of recording technique, so does vestibular examination utilizing nystagmus Thus, ENG has become one of the most important measures in vestibular investigation and examination

Spontaneous nystagmus which is suspected to exist from patients history or from observation with Frenzel's spectacles is checked by ENG Spontaneous vestibular nystagmus is unique in that it appears most distinctly with the eyes closed or covered (Fig 1) Invisible nystagmus can be recorded by ENG When existence of nystagmus is doubtful, "adequate measures" to activate nystagmus are sometimes useful, as heretofore mentioned

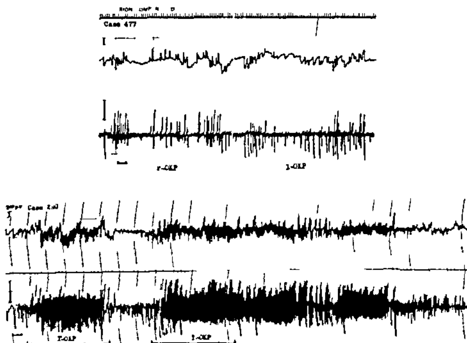


Fig 5 Optokinetic patterns obtained in spontaneous nystagmus of central origin (A) and those of ocular origin (B) Note that the characteristic patterns are entirely different from those of peripheral vestibular nystagmus

4) Optokinetic pattern test (OKP test)

Optokinetic pattern test was introduced by Suzuki, one of the authors as a clinically available test (Suzuki 1962) Optokinetic patterns of spontaneous nystagmus of central or ocular origin are entirely different from those of spontaneous vestibular nystagmus (Fig 5) OKP is, therefore diagnostically important and contributory for differentiation of spontaneous nystagmus (Suzuki 1962)

Another important factor of the optokinetic pattern test is that it enables quantitative assessment of the intensity of spontaneous vestibular

nystagmus Spontaneous vestibular nystagmus influences optokinetic fusion limit, i e, elevates the fusion limit of optokinetic nystagmus provoked towards a particular direction (left or right) of spontaneous nystagmus, while it lowers that in the opposite direction Spontaneous nystagmus is thus intensified by optokinetic stimulation towards the direction of spontaneous nystagmus in proportion to its intensity, while reduced in the opposite direction, OKP is modified to be asymmetric and existence of spontaneous nystagmus is schematically visualized The intensity of spontaneous nystagmus is assessed by measuring the area of the slow phase of eye speed recording of OKP, corresponding to total amplitude of optokinetic nystagmus induced on spontaneous nystagmus An OKP test conveniently applied for quantitative assessment of restoration after acute vestibular disturbance is as shown in Fig 6 (Komatsuzaki 1961)

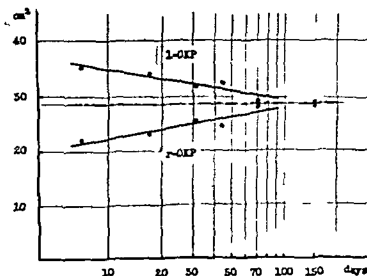


Fig 6

... thmic scale
ordinate area of the slow phase of OKP in cm²

5) Mobilization (activation) of spontaneous nystagmus through optokinetic stimulation

Optokinetic stimulation is utilized for augumentation or mobilization of latent spontaneous nystagmus Spontaneous nystagmus with extremely small amplitude is hardly detectable behind Frenzel s spectacles, or hardly recognizable with ENG or from OKP test

In the event of the above mentioned four procedures failing to demonstrate spontaneous nystagmus being suspected, after nystagmus following optokinetic stimulation is examined electronystagmographically (Fig 4) Optokinetic stimulation is given acceleratively, based on the fundamental experiment showing that after-nystagmus is more actively provoked by stimulated acceleratory rotation of the drum than by that of a constant rotatory speed (Mizukoshi 1961) Spontaneous nystagmus is markedly augumented by optokinetic stimulation to the direction of the quick phase of spontaneous nystagmus When marked difference of after nystagmus is demonstrated between right and left rotations in cases where spontaneous nystagmus is suspected but not distinct, the condition is regarded to correspond with directional preponderance which is of highly diagnostic importance

ZUSAMMENFASSUNG

In der Prufung der Patienten, die vom Schwindel bzw Gleichgewichtsstorung gelitten sind, sind Stress und Spontannystagmus zu beweisen

Die Ordnung der angewandten Prufung des Spontannystagmus beruht auf der Erfahrung, die von den bisher in unserer Klinik untersuchten Patienten gewonnen ist

Die angewandte Ordnung ist folgend

1) Prufung des Spontannystagmus mit geoffneten Augen,
2) Prufung des Spontannystagmus mit Frenzel's Leuchtbrille,
3) Prufung des Spontannystagmus im Gebrauch mit Elektronystagmographie,
4) "Optokinetic Pattern Test" (OKP-test),
5) Mobilization des Spontannystagmus durch optokinetischen Reiz

Die erste vier obengenannten Methoden haben jeder fur sich eine Wichtigkeit und sollen immer als genotigte Untersuchung in allen Fallen des Spontannystagmus oder der Gleichgewichtsstorung ausgefuhrt werden

Die dritten, vierten und funften Prufungen werden durch die electronystagmographische Registrierung ausgefuhrt Es ist keine Übertreibung, wenn ich ihre klinischen Eigentumlichkeiten betone

"Optokinetic Pattern Test", das ist in unserer Klinik ausgefuhrt worden bringt zu uns eine der grossten Taugbarkeit fur beide der Differenzierung des Spontannystagmus und quantitative Analyse der Starke der spontanen vestibularen Nystagmus

Die letzte Prufung, in der optokinetischer Nach nystagmus zwischen den links und rechts vergleichen wird, wird besonders in solchen Fallen ausgefuhrt, dass das Dasein des Spontannystagmus verdachtig ist, aber in vier obengenannten Methoden nicht sicherlich beweisen werden kann

ACKNOWLEDGEMENT

The authors wish to express sincere gratitude to Professor Frederick G H Smith for his kind advice in correcting the manuscript

REFERENCES

ASCHAN, G, BERGSTEDT, M and STAHLE, J, 1956 Nystagmography, recording of nystagmus in clinical neuro otological examinations *Acta Oto laryng Suppl 129*
DIX, M R and HALLPIKE, C S, 1952 The pathology, symptomatology & diagnosis of certain disorders of vestibular system *Ann Otol Rhin and Laryng 61*, 987
FRENZEL, H, 1955 *Spontan und Provokationsnystagmus als Krankheitssymptom* Springer Verlag
JONGKEES, L B W, 1960 Some remarks on the function of the vestibular organ *J Laryng 74* 511
KOMATSUZAKI, A, SUZUKI, J, TOKUMASU K, SAKATA E and GOTO, K, 1961 Surgical treatment of Meniere's disease, Part II *Otolaryng (Tokyo) 33* 10
LINDSAY J R, 1951 Postural vertigo and positional nystagmus *Ann Otol Rhin and Laryng, 60*, 1134
MIZUKOSHI K, 1961 Some observations on the optokinetic nystagmus *Jap Jour Otol* 64, 23, 207
MORIMOTO M, 1955 Otoneurological study on the brain tumor *Acta Med Biol 3*, 243
NYLÉN C O, 1931 A clinical study on positional nystagmus in cases of brain tumour *Acta Oto laryng Suppl 15*
SAKATA E Studium uber den beider Wechselung der Kopf-oder Korperlage hervor gerufenen Nystagmus Ein Vorschlag der Untersuchungsmethode und die Betrachtung uber interessante klinische Falle *Arch Ohren usu Heilk u. Z Hals usu Heilk* (in press)
——— Differenzial diagnostische Bedeutung von der Nystagmus *Arch Ohren usu Heilk u z Hals usw Heilk* (in press)
STENGER H H, 1955 Über Lagenystagmus unter besonderer Berucksichtigung des gegenlaufigen transitorischen Provokationsnystagmus bei Lagewechsel in der Sagittalebene, *Arch Ohren usu Heilk u Z Hals usu Heilk 168*, 220
SUZUKI J and KOMATSUZAKI A, 1962 Clinical application of optokinetic nystagmus, optokinetic pattern test, *Acta Oto laryng 54* 49

Motofujicho Hongo, Tokyo

QUANTITATIVE OBSERVATION OF VESTIBULAR REACTIONS IN THE REVOLVING ROOM

YUTAKA TSUIKI, SHUICHI KATAGIRI, JIRO HOZAWA, MIYUKI KAKUTA, MASAMI USAMI and KAZUKO SATO
Sendai, Japan

From The Department of Oto Rhino Laryngology, (Emeritus Prof Y. Tsuiki, Head Prof S Katagiri) Tohoku University School of Medicine, Sendai

The authors bilt a revolving room, in order to investigate quantitative interaction between angular acceleration and labyrinthine function The subject was made to stand at the centre of this room, with his head bent 30° forwards, and his eyes open
The various responses induced by rotatory stimulation, namely, nystagmus, turning sensation, change of respiration and head movement were simultaneously observed by electric and photoelectric methods As a result the authors found that it is possible to diagnose not only vertigo but also motion sickness and to test the fitness of pilots

Many experiments on nystagmus during rotation have been reported Nystagmus alone is not a vestibular response included by angular acceleration Therefore, the authors would like discuss here the following phenomena

i) nystagmus
ii) turning sensation
iii) change of respiration
and
iv) head movement

1 A device of revolving room for quantitative observation of vestibular function

A revolving room was built in order to investigate quantitative interaction between angular acceleration and labyrinthine function

This revolving room was made to rotate in a such a way as to eliminate non-labyrinthine stimuli such as light, sound and air movement, therefore, only angular acceleration is applied to the subject

This device could produce 12—80°/sec rotation velocity and could also selectively produce any angular acceleration rate between 0 13—7 6°/sec²

2 *Methods of observation of vestibular reactions*

1) The subject was made to stand at the centre of this room, with his head bent 30° forwards and his eyes open (Fig 1)

F g Ge e a w o he s g oom

The e o g oom | o ro pa
3 Amp E N G etc | 4 Pen-osc g ph
5 E ec od box | 6 rc ry pool and o ac
7 Sh dcurd | 8 In pt
9 N st gmus E N C | C Pho oce
L gh | 2 E ec rod Resp a ion
3 S gna key | 4 O pu

2) The responses were observed indirectly by electric and photo electric methods namely the nystagmus by electro-nystagmography (E N G), the turning sensation by a signal given by the subject, the change of respiration by electrorespirography and the head movement by tracing the movement of a photoelectric bulb (Fig 1)

3) i) Angular acceleration stimuli of 2°/sec², 1°/sec², or 6°/sec² were applied for ten seconds and the latency of various reactions induced by rotatory stimulation was measured

ii) After the subject was rotated at constant velocity of 20°/sec, 30°/sec or 60°/sec the rotation was suddenly stopped and the duration of various reactions induced by postrotatory stimulation was measured (Fig 2)

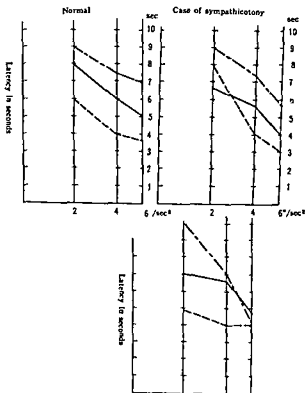
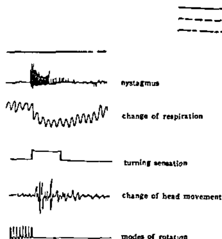


Fig 5 Latency of perrotatory reactions

perrotatory nystagmus
change of resp rat on
change of head mo e nent



F g 6 Durat on of postrot tory reaction (60 /sec)

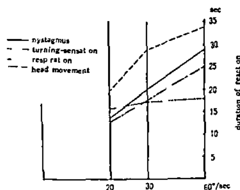


Fig 7 Duration of various vest bular react ons nduced by acceleration (average of normal 28 cases)

2) Duration of response

As shown in Fig 6, the duration of response is not always the same Fig 7 is an average pattern of 28 normal cases All the patterns indicate an upward curve to the right The turning sensation curve shows

least elevation and therefore is crossed by the curve of nystagmus and head movement The other curves are approximately parallel The duration of respiratory change in the case of normal person is prolonged more than nystagmus and the head movement disappears earlier than nystagmus

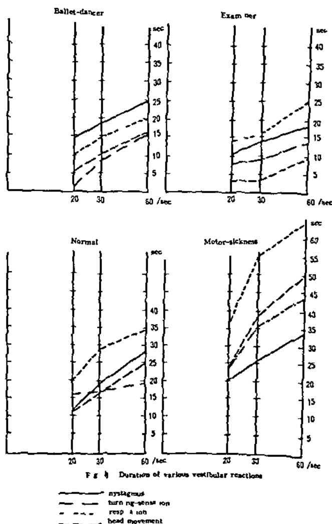


Fig 8 Duration of various vestibular reactions

But as shown in Fig 8 the duration of reactions are prolonged in the case of motion sickness and moreover the inclination of its curve is steepened and the duration of respiratory change is remarkably prolonged These differences from reactions of a normal person are clearly observed

Subjects inured to examination in the revolving room and especially ballet-dancers lose other phenomena earlier than nystagmus

These findings show marked contrast to those in motion sickness Even when no notable result can be obtained from the observation of

vertigo, lethargy etc), sensory and motor disorders and other E N T disorders Secondly, we have examined the alteration of blood pressure and pulse count of the asked subjects We have measured the highest blood pressure when they lie in bed quietly and then also the highest pressure when they have risen (the time of this measurements is five minutes after rising) When the figures of these measurements have been applied to the following formula the changing indexes of blood pressure have been marked as from 0 to 0 190 We have determined the average figure of these changing indexes 0 078 as autonomic stable (normal) and more than 0 09 as autonomic unstable (abnormal) In regard to the pulse count, the same formula has been applied where we have judged the changing indexes of more than 0 12 as autonomic unstable

$$\textit{Changing index of blood pressure} = \frac{\textit{Highest blood pressure when standing} - \textit{Highest blood pressure when lying}}{\textit{Highest blood pressure when lying}}$$

$$\textit{Changing index of pulse count} = \frac{\textit{Pulse count when standing} - \textit{Pulse count when lying}}{\textit{Pulse count when lying}}$$

Considering the above points, (asking diagnosis, the changing index of blood pressure and pulse count) we have classified them into two, those in a state of stable, and unstable groups of autonomic nervous system Of these two groups, we have made the following so called vestibular labyrinthine function test

Vertical writing with covered eyes As seen in the *Chart No 1* which shows the degree of deviation of writing from a vertical line, the members of the stable group have made a trifling deviations, while those of the unstable group have shown remarkable ones On examining the post rotatory deviation when subjects have been revolved with cupulometry method, the difference between these two groups has appeared more remarkably

Chart No 2

When in this graphic chart the changing indexes of blood pressure have been indicated on X line and those of pulse count on Y line, fixing 0 09 and 0 12 respectively as limitation of the figures regarded as a stable state of autonomic nervous system, the deviations within 6° of vertical writing can be considered as normal, while those which have excessed over 7° (unstable subjects) are seen scattered in the area regarded as unstable

Chart No 1 Angle of deviation in writing with eyes covered and the changing index of pulse count and blood pressure

		stable group	unstable group	total
number of cases		26 (54 1%)	22 (45 9%)	48 (100%)
angle of deviation	variation range	0°—6°	0°—9°	0°—9°
	mean	2.3°	4 3°	2.9°
post rotatory deviation	variation range	3.5°—12°	4°—18°	3.5°—18°
	mean	6 0°	11.9°	10.5°
	no of cases	22	14	36

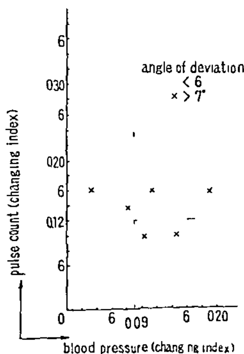


Chart No 3

Revolving a subject with accelerated high speed once a round who has stable nerve, we have observed his post rotatory nystagmus The rotation is counter-clockwise and Barany method In this test, the nystagmus we have observed appears emphatically at the beginning of the rotation and then it gradually becomes small and intervals long After the stop of rotation the same phenomen occures in the reverse direc tion The pulse wave (finger plethysmogram) recorded simultaneously with the rotation does not show an intensive influence. This is quite an ordinary well known phenomenon

its frequency But only small influences can be seen on any optic or central nystagmus by the respiratory suspension

From this fact, we can infer that the nystagmus is also subjected to the respiratory influences

On further experiments we have obtained the followings

(1) Rotations with angular acceleration of 1 2°/sec² for autonomic stable group did not induce nystagmus after two revolutions Many of the subjects showed nystagmus during the third revolution On the other hand the change in pulse wave begins approximately from 10 to 20 seconds after the start of rotation

(2) In regard to the autonomic unstable subjects, nystagmus appear from 2 to 4 seconds after the start of rotation, while the change in pulse wave begins from 12 to 18 seconds after the start Namely, the change in pulse wave is noticed in both groups by rotation with certain angular velocity (approx from 10° to 12°/sec) While the appearance of nystagmus is very changeable according to the state of autonomic nervous system of the time, stable or unstable

(3) *In the case when the rotation starts with small angular accelera* tion of 0 8°/sec , and stop on the moment when the speed has got to 30°/sec , the average duration of post rotatory nystagmus has been 14 6 sec for the stable group, while for the unstable group it has been 30 6 sec

(4) In the case when the rotation was made ten times with a constant angular speed of 30°/sec , the average duration of post-rotatory nystagmus has been 13 9 sec for those stable group, while for those unstable group it has been 28 1 sec

(5) In rotation test with angular speed of 60°/sec , which is believed to be a suitable stimulus to cause a clear response, the average duration of the post rotatory nystagmus has been prolonged to some extent We, however, prefer smaller angular speed of less than 60°/sec , which might be rather exact and effective to observe a proper response of vestibulum

But when we made rotation tests at random with constant angular speed of 30°/sec twice and ten times, and then with constant angular speed of 60°/sec also twice and ten times, the durations of the post-rotatory nystagmus of the same subjects were quite various and different in each cases Even in the stable group it was difficult to find out a certain tendency or a rule Especially as for the unstable group the duration of nystagmus showed no rule, having no relation both to the angular speed and the number of rotation times

ZUSAMMENFASSUNG

Wenn wir die Drehprufung zur Prufung der Labyrinthfunktion anwenden, mussen wir beachten, dass die Reaktion je nach dem Zustand des autonomen Nervensystems der Versuchspersonen verschieden ist

Obgleich die Versuchsperson gesund sein kann, beeinflusst die individuelle Stabilitat und Labilitat des autonomen Nervensystems bei der Prufung in hohem Masse die Resultate In der Tat, die Mehrzahl der autonomisch labilen Versuchspersonen sind parasympatisch uberempfindlich Nur wenn das autonome Nervensystem der Versuchsperson stabil ist und ein schwacher Drehreiz angewandt wird, ist die Dauer des Drehnachnystagmus ein Merkmal fur die Beurteilung der Vestibularfunktion

REFERENCES

The functional tests

BÉKESY, V G, 1955 Subjective cupulometry, *Arch Otolaryng 61* 16

FUKUDA, T, 1959 The stepping test, *Acta oto-laryng 50* 95

FUKUDA, T, 1959 Vertical writing with eyes covered, *Acta oto-laryng 50* 26

HOSEKO, S, KAWAKUBO J and TOGANOW Y 1957 Deviation on the sagittal (vertical) writing with closed eyes (The drift reaction of the arms) of the normal people *Jap Jour Otol Tokyo, 60*, 1096

SCHMALTZ, G, 1932 The physical phenomena occurring in the semicircular canals during rotatory and thermic stimulation, *Proc Roy Soc Med 25* 359

STAHLE, J, 1958 ENG in the caloric and rotatory tests, *Acta oto-laryng Suppl 137*

STAHLE, J, 1957 ENG in the rotatory test, a study in a series of normal individuals, *Acta Soc. Med. Upsalien 62*

WODAK, E, 1957 The dissociations of the different vestibular reflexes and their diagnostic significance, *Pract oto-rhino-laryng* 19, 587

Plethysmography

BUCH G E, 1954 *Digital plethysmography* Grune & Stratton N Y

NAGASHIMA C, 1957 *Nervous control of human blood vessels* Tokyo University Press

Autonomic nervous system

BERGMANN G V, 1928 Funktionelle Pathologie *Zbl Klin Med. 18* 90

EPPINGER, M, and HESS L, 1909 Vagotonia *Zbl Klin. Med 67* 345

NEUBERGER F, & SCHMIDT R, 1960 Uber der vegetativen Halbseitentonus und die seitengleiche Spezifitat peripharer vestibularisreize *Pract oto-rhino laryng 22*, 365

PREBER, L, 1958 Vegetative reaction in caloric and rotatory test. *Acta oto-laryng Suppl 144*

1-chome Kashiwagi Shinjuku Tokyo

AN APPLICATION OF STRAIN GAGE-TYPE INSTRUMENTS TO THE ANALYSIS OF THE EQUILIBRIUM FUNCTION IN HUMAN SUBJECTS AND ANIMALS

MANABI HINOKI and MASAAKI KITAHARA
Morioka, Japan

From the Department of Otorhinolaryngology (Head Prof M Hinoki, M D) Iwate Medical College, Morioka

First of all introducing the principles of the strain gage and also the characteristics of the strain gage type instruments hitherto described, two of the instruments specially devised by the authors for the purpose of precise recording of the body movements in human subjects or animals under various conditions, such as labyrinthine or visual stimulation, have been presented in detail Using an unbonded strain-gage-type linear accelerometer or the two newly devised strain gage type instruments, together with either electronystagmograph or electromyograph, the equilibrium function in human subjects or animals has been successfully analysed

INTRODUCTION

In order to analyse the equilibrium function in human subjects or animals, it is, as a rule, necessary accurately to observe and record the voluntary as well as reflective movements of the eyeball and body However, while the recording of eye movements is presently an easy thing, since such can now be precisely recorded by the electronystagmograph (ENG), that of body movements is still considered to be rather difficult, although several methods by various researchers have already been described

To improve the method of recording body movements, a kind of strain gage-type instrument was introduced into this field of study as an accelerometer in 1959 (Kitahara) By an inventive use of various stain-gage type instruments, the authors have succeeded in precisely measuring the body movements, the acceleration caused by parallel swinging, rotation, and the like

In this paper the authors explain the characteristics of this apparatus and then describe the utilitarian value of this method for eld of study

STRAIN GAGE-TYPE INSTRUMENT

The strain gage consists of a short length of w
to the material being tested The strain occurr
rise to a change of electrical resistance which is a

measuring circuit used is a Wheatston Bridge which is activated by an oscillator The output, a sine wave of oscillator frequency modulated by the sigal wave, is amplified and rectified by a copper oxide rectifier before recording (Dobie 1950) There are various types of strain-gage transducers, however, the ones used in the research studies of equilibrium function are such as follows·

(A) Accelerometer

i) Classification of accelerometers

The accelerometers used in this investigation are composed of strain-gages which are closely connected to the inertia mass These gages construct a Wheatston Bridge, so that the output parallel to the acceleration is obtained The accelerometer of this type has a higher natural frequency and sensitivity for its size than the other type of accelerometers, e g, mechanical type, induction type accelerometer, etc (Egmond 1952) The accelerometers used in this investigation are classified into two types according to their construction One is an unbonded strain gage type accelerometer, the other is a (bonded) strain-gage type accelerometer

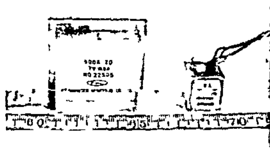


Fig 1 An unbonded strain gage-type linear accelerometer (left) and a (bonded) strain gage type linear accelerometer (right)

The accelerometer of the former type consists of two pairs of stretched wires which suspend an inertia mass so that under acceleration the tension of one pair of wires is increased while that of the other is decreased The accelerometer of the latter type consists of wire pasted on a base which is held in close relation to the inertia mass, so that any strain caused by the mass can be transmitted to the gage The accelerometer can also be classified as two other types i e, the linear accelerometer and the angular type In the former accelerometer construction is devised so that a small metallic weight enclosed in the apparatus, works as an inertia mass during the linear movements of the apparatus while in the latter accelerometer, the shaft of the apparatus plays a part in the inertia mass during the rotation of the apparatus

ii) Selection of an accelerometer

To measure greater acceleration, it is recommended that an accelerometer with a high natural frequency be used, because almost all the time greater acceleration is caused by rapidly moving materials Conversely, to measure smaller acceleration, it is necessary to use the accelerometer with a low natural frequency, because this kind of accelerometer shows high sensitivity Generally, the unbonded strain gage-type accelerometer has a higher natural frequency and sensitivity than the (bonded) strain-gage type accelerometer Therefore, the unbonded one is the one suitable for registering and measuring the acceleration component of movements of the human body or linear acceleration caused by both parallel and vertical swinging The angular acceleration caused by rotation is registered by an angular accelerometer But the angular acceleration may be registered also by the linear accelerometer through registration of the tangential acceleration of a point some distance from the rotation center In order to record the angular acceleration of the rotation chair, in most cases, it is necessary to improve the rotation chair, e g, the diminution of motor vibration, etc At the present time, the (bonded) strain-gage-type accelerometer has a less natural frequency and sensitivity than the former Therefore, the accelerometer of this type is undesirable for the registration of fine acceleration which occupies an important place in our present day labyrinthine examination A peculiarity of the (bonded) strain gage-type accelerometer is that it is very small and light, so it can be used for the registration of the head movements of small animals

iii) Calibration of acceleration

Calibration of acceleration is an important procedure in the laboratory By using the procedures which are mentioned below, the authors made calibration of the acceleration which was used in the experiments described below In the case of measuring acceleration of less than 1 0 g, calibration was made by estimating a component force of gravity charged upon the accelerometer which was inclined in a specific direction and at various degrees, whereas in the case of measuring acceleration of more than 1 0 g, the centrifugal force, caused by the rotation instead of by gravity, was used in the calibration In the latter case it is easier and more accurate to change the angle caused by the direction of the sensitivity of the accelerometer and that of centrifugal force, than to change the rotation velocity

(B) Strain-gage

To record the position of the head in relation to the trunk, or the position of the four extremities in relation to the trunk, etc, the strain-gage is most useful There are many ways to transfer body movements to the gage However in the case of applying the strain gage type instruments to the subject, it is most important to keep the load on the body as light as possible

(C) Load cell

In 1958, H Shugyo reported the physiological sway of the center of gravity and postural stability Later he introduced a kind of load cell as an experiment, and succeeded in getting the plane projection of the center of gravity of the human body on the oscilloscope screen

OBSERVATIONS

From the present study, which was undertaken by using the strain gage-type apparatus to analyse the characteristics of the head and body movements and those of the motion of instruments such as the parallel swing, the authors present the following observations

In Fig 2 are presented the registered movements of the eye and head in cases where the head is actively rotating against the trunk around the longitudinal axis of the body, with the eves being open to the right and left (Hinoki 1961)

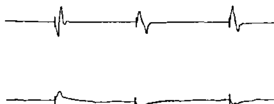


Fig 2 The registrogram of the eye and head movements of a 22-year old man with normal healthy ears, the head is actively rotating against the trunk around the longitudinal axis of the body The upper line indicates the head movements, while the lower, the eye movements

The upper line is the registrogram of the head movements and the lower one is that of the eye movements In order to register the eye and head movements, the authors used the ENG and the unbonded strain gage type linear accelerometer, respectively In the case of registration of the head movements, the accelerometer was attached to the middle of the forehead of the subject so that the acceleration from side to side could be measured In the procedure mentioned above, *the tangential acceleration*, which is in direct proportion to the angular acceleration caused by the head rotation, can be recorded The upward spiking curves indicate the angular acceleration to the left, which occurs both in the beginning of head rotation to the left or in the end of head rotation to the right, whereas the downward spiking curves indicate the angular acceleration to the right, which occurs both in the beginning of head rotation to the right or in the end of head rotation to the left

Using the instruments mentioned, the authors *were able to confirm* that there is a close relationship between the eye and head movements in the case of head rotation This relationship may be classified into seven

patterns according to the direction and time of the onset of the eye and head movements at the moment of the beginning of head rotation Furthermore, it was noted that each of the patterns is closely related to the state of the equilibrium function of the subject investigated

In this case, with the equilibrium function of the subject considered to be in a state of sufficient development, the eye movements coincide, in direction and in time of onset, with the head movements at the moment of the beginning of the head rotation This pattern was able to be observed in the majority of normal healthy adults

In Fig 3, the optokinetic eye and head nystagmus of a guinea pig are presented

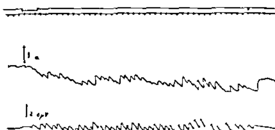


Fig 3 The optokinetic eye and head nystagmus of a guinea pig with normal ears The optokinetic stimuli, produced by the rotation of a large cylinder (Fukuda 1957), were regulary applied to the animal for twenty seconds at the rate of one rotation per forty seconds The upper line indicates the optokinetic head nystagmus, while the lower, the optokinetic eye nystagmus

In order to register the head movements of a guinea pig, the authors devised the apparatus shown in Fig 4 As observed in Fig 4, this apparatus consists of three parts as follows The first part is composed of a strain-gage pasted on thin elastic plate, one edge of which is fastened to a supporter and the other edge of which is connected with a fine steel pole The second part consists of a fine steel pole, 1 mm in diameter, 20 cm in length, which joins the thin elastic plate, mentioned above, to a 3 cm long shaft This shaft comprises the third part, one edge of which is a ring 1 1 mm wide in diameter, and the other edge is fastened to the head of the animal investigated with an adhesive plaster The fine steel pole described above is inserted into the ring so as to have recorded as exclusively as possible only the head movements from side to side

After the above preparations have been completed, the head nystagmus of the animal which is produced by several kinds of stimuli, e g, visual and labyrinthine, is conveyed to the thin elastic plate through the fine steel pole, and thus the strain on the gage is effected The authors were able to establish that the amount of the strain on the gage was in direct proportion to the distance of the head movements of the animal, and furthermore, that the head movements of the animal were not effected by application of this apparatus Therefore, the authors would like to recommend this method as a very useful one in measuring the head nystagmus of small animals

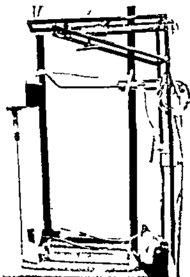


Fig 4 The strain gage type instrument devised by the authors to record the head nystagmus of a small animal such as a guinea pig.

Using the instruments described above the authors were able to confirm that the eye and head nystagmus caused by the visual stimuli are closely related to each other, the relationship being divided into various types of pat terns Furthermore, in the authors' opinion, the types of the patterns depend primarily upon the state of the equilibrium funct on of the animals

In the case at hand, the equilibrium function of the animal was relatively well developed due to the repetition of the visual stimulation The eye and head nystagmus provoked by visual stimuli, were coincident in direction and time with the onset and duration of movement during the time of the head nystagmus with the exception that the eye nystagmus occurred more frequently than the head nystagmus in the early stage of the visual stimulation

In Fig 5a b are shown the cephalograms obtained in Fukuda's step ping test (Fukuda 1959)

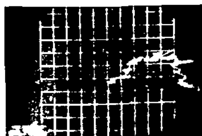


Fig 5a The cephalogram taken while the subject is stepping up and down and shows the registered reactions as they appeared from ten to forty seconds after syringing the right ear with 10 cc of water at 20° C



Fig 5b The cephalogram taken while the subject is step ping up and down four minutes to four and a half minutes after syr inging the right ear with 10 cc of water at 20° C

In order to record the head movements of the subject while stepping up and down, the authors made the apparatus described below In this apparatus a pair of thin elastic plates, on each of which is pasted a strain-gage, are joined together at a right angle Each of the strain gage apparatus consists of two parts, i e, the strain gage pasted on the thin elastic plate and the fine elastic string which joins the thin elastic plate to a band attached to the subject's head with slight tension In this case, the supporter fastened to the thin elastic plate is fixed at the ceiling, and an elastic tape of about 2 m is used as the string After completion of the above mentioned preparations, the change in position of the subject's head, which change is caused by stepping up and down, is conveyed to the fine elastic plates through the fine elastic tape, and thus the strain on the gages is effected According to the characteristics of above mentioned apparatus, the amount of strain on one gage is considered to be in direct proportion to the amount of change in the position of the head as registered on the back and forth axis (Y axis in Fig 6), whereas in the case of the other gage, the amount of strain is considered to be displayed in a similar manner on the side to side axis (X axis in Fig 6) Therefore, when the output of each gage is connected with the Brown tube type oscilloscope as shown in Fig 6, the head movements of the subject can be observed as movements of the pip on the screen

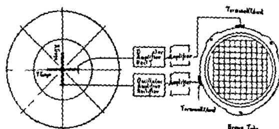


Fig 6 The block diagram of the strain gage type instrument devised by the authors in order to record the cephalogram of a subject stepping up and down

In Fig 5a is shown the cephalogram taken while the subject is stepping up and down, and shows the registered reactions as they appeared from ten to forty seconds after syringing the right ear with 10 cc of water at 20°C Fig 5b shows the cephalogram taken while the subject is stepping up and down, four minutes to four and a half minutes after syringing the same ear with 10 cc of water at 20°C In the former, the head movements are accompanied by marked ataxia, while in the latter, the head movements are performed smoothly In correlation with this fact, it has been further noted that in the two cases of the up and down stepping, the direction of the head deviation and rotation in both cases, is directly opposite The authors taking Fukuda's classification into consideration, assume that the former case corresponds to the stage of disturbance of the labyrinthine reflex while the latter, to the stage of coordination It is worthy of note that the characteristics of both cases can be clearly recorded by this apparatus

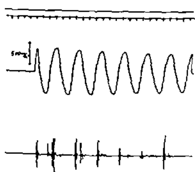


Fig 7 The registrogram of the motion of a swing and the EMG of a chicken's tail muscle (m levator coccygicus) while the chicken is being swung on a swing The upper line indicates the linear acceleration of the swinging motion, while the spikes of the lower line indicate the muscular discharges from a chicken's tail

In Fig 7 are shown the motion of a swing and the electromyogram (EMG) of a chicken s tail muscle (m. levator coccygicus) while swinging The upper line indicates the linear acceleration of the motion of a swing, while the spikes of the bottom line indicate the muscular discharges from the chicken's tail To register the linear acceleration of the motion of the swing, the authors used the unbonded strain gage-type linear accelerometer attached to the swing As is shown in Fig 7, the motion of the swing was precisely registered by this method, and it was possible to observe precisely the relationship between the motion of the swing and the muscular discharges in the chicken's tail caused by the swinging motion

It has been found by the authors that the muscular discharges in the chicken's tail while swinging are of manifold patterns, which are in accord with the state of their equilibrium function (Fukuda 1961) Especially worthy of note is the fact that the muscular discharges in a trained chicken, as compared with those of an untrained one, were markedly different in their magnitude, frequency and the place of occurrence within the entire cycle of the swing In the case at hand (Fig 7), the equilibrium function was noted to become well developed by daily repetition of the pendulum-like motion In a well trained chicken the muscular discharges during the entire cycle of the swinging motion were not pronounced, being infrequent and appearing chiefly at the top of the sine wave, which point corresponds to the limit of the backward returning motion of the swing.

COMMENTS AND ANALYSIS

The reflex patterns of the equilibrium system, especially those of the spinal reflex caused by various kinds of stimulation, e g, visual and labyrinthine, have already been examined by many researchers using such various methods as cephalography, cinematography, photo-electric method, electromyography, etc However it is generally accepted that these methods have some disadvantages with regard to recording the movements of the body

With respect to cephalography, the movements of the body (especially those of the head) can only be recorded within a certain limit, due to the characteristics of this method That is to say, in this method the movements of the body are recorded as a cephalogram on the surface of a piece of paper covered with soot, with the needle of the apparatus being attached to the subject's head Thus the movements of the body during various kinds of active movements, such as active rotation, jumping, walking, etc, can not be registered by this method

With respect to photo electric methods, the recording of body movements must be carried out in a dark room, due to the characteristics of those methods

Therefore, various kinds of experiments requiring the subject's visual aid can not be performed by the methods mentioned above

In recent years special attention has been given to investigations in electromyography, and by using this method many important reports have been presented in the field of study which treats of the equilibrium function in human subjects or animals Electromyography is an excellent method for analysing the actions of each muscle fiber and for determining the characteristics of the reflexes caused by various kinds of stimulation However, even this procedure can not be regarded as ideal for recording the movements of the body, for, when the needle electrode is applied to a muscle, the defense reaction acts inevitably against the needle, hindering the observation of the equilibrium function of the body Especially recording of the actions of a muscle during relatively strenuous, active movements of the body, such as active rotation jumping etc, is considerably disturbed by the voluntary and reflective defense reactions caused by this procedure

It is of interest to note what role strain gage type instruments can play in this field of study since, by using strain gage type instruments, the disadvantages mentioned above are seen to be overcome

The principle of the strain gage was discovered by Load Kelvin in 1856 Strain gages have been applied not only in measuring the strain and stress of materials under test, but also in constructing various kinds of transducers in technology

Recently strain gage type instruments have also been introduced into the field of medical research and some investigations have been carried out with the use of these instruments e g, blood and intra ocular pressure has been able to be measured and also masticatory force has been able to be examined with the use of these instruments (Katsuki, Sasamoto 1957) As for the labyrinth as has already been pointed out by Fukuda (1957), this organ should play an important role during the active movements of the body Therefore research in the method of recording movements in such cases must be earnestly and actively promoted However, it would seem investigations of this type have not been sufficiently performed

The first object of the authors' investigations was to solve the difficulties in recording active movements of the body, and for this purpose, strain gage-type instruments were applied One of the authors, Kitahara, introduced in 1959 an unbonded strain gage type accelerometer into the field of study of the equilibrium function (Kitahara 1959) He succeeded in recording the precise head movements of the human body Furthermore, in keeping with the purpose of each experiment previously described, the authors devised two new kinds of strain gage type instruments, and by using these instruments, were successful in precisely recording of the above mentioned movements (see Figs 3, 5)

From the observations described above, the authors wish to emphasize the suitability of their procedure for the purpose at hand The success in recording the movements can be ascertained from the following facts

1) Following the above procedure, various kinds of factors affecting the equilibrium function of the body, such as acceleration, pressure, and other stresses, (e g , the deviation of the head in relation to the trunk, and the deviation of the extremities in relation to the trunk, etc) can be precisely registered

2) Furthermore, it is worthy of note that, when these instruments are used, their measuring range and sensitivity can be easily controlled, and, in addition, that they are small in size, light in weight, and do not give any pain during application Therefore, this procedure is regarded as almost ideal for recording the movements of the body

In the authors' opinion, one of the essential problems to be solved in the study of the equilibrium function in human subjects or animals, is that the eye and body movements should be recorded simultaneously, and that then the relationship between them should be analysed With respect to the eyes, the movements have been precisely recorded by the ENG The movement of the body, however, have not yet been recorded so precisely as those of the eyes, though various methods have been tried by various researchers

The second object of the authors' investigation was to learn what role strain gage type instruments could play in solving the above mentioned problem. As observed in Fig 3, the nystagmus, caused by the optokinetic stimuli, was able to be precisely measured, that is, the eye nystagmus measured by the ENG and the head nystagmus by the strain gage type instrument Also, as observed in Fig 2, the eye and head movements, in cases of active head rotation were registered precisely with the use of the ENG and the unbonded strain gage-type linear accelerometer

From these observations, the authors would like to state that the relationship between the eye and body movements, e g , the head movements, was precisely analysed for the first time with the use of both strain gage-type instruments and the ENG

Furthermore, the other essential problem to be solved in the study of the equilibrium function in human subjects or animals is the fact that the

precise amount of stimulation and reflexes caused thereby, should be simultaneously recorded, and the relationship between them should be precisely analysed. However, the recording methods of the present day, as far as those which pertain to acceleration are concerned, are not always sufficient for such recording. Precise and convenient recording methods of acceleration have not yet been devised in the field of study which treats of the equilibrium function in human subjects or animals. Accordingly, the third object of the authors' investigation was to learn what role the strain-gage-type instruments could play in solving this problem. As observed in Fig. 7, the linear acceleration caused by the swinging motion was precisely recorded during the time of the swinging, and it was possible to analyse clearly the relationship between the muscular discharges in the chicken's tail while on the swing and the linear acceleration caused by the swinging motion. From these observations it is clear that the strain-gage-type instruments can play an important role in solving the problem described above.

Finally, out of their experience the authors would like to offer a few words of guidance relative to the use of strain-gage type instruments.

1) When strain gage-type instruments are used beyond their limits in measuring the subjects' movements, experimental errors inevitably occur. Therefore, it is important to estimate the frequency and magnitude of the subjects' movements before attempting to measure the characteristics of such movements, and then select a strain gage type instrument suitable for the case at hand.

2) As is generally accepted, when strain gage type instruments are used, the temperature and humidity of the atmosphere effect the results of experiments to some extent. In the authors' experiments, however, these factors affected the results to only a slight extent and can, we feel, therefore be disregarded.

In short, strain gage type instruments pose an excellent utilitarian value in the field of study which treats of the equilibrium function in human subjects or animals. Especially it is worth noting that they show many excellent characteristics in analysing body movements which have not heretofore been investigated in as much detail as eye movements. Furthermore, it should be stressed here that the various strain gage-type instruments previously mentioned are all presently easily accessible, thus raising the utilitarian value of the instruments even more.

ZUSAMMENFASSUNG

Zuerst werden das Prinzip des "strain-gage" und die Eigenschaften der Instrument von "strain gage type" vorgelegt. Dann werden die zwei Instrumente die besonders, um die Zustande der Korperbewegungen von Menschen und Tiere die in den verschiedenen Umstanden, Labyrinth, Gesichtanreizung usw. gestellt sind, genau zuverzeichnen, von den Verfas-

sern erfunden sind, in die Einzelheiten dargestellt Angewendet mit Elektonystagmograph oder Elektomyograph, haben die "unbonded strain-gage-type linear accelerometer" und diese zwei neu erfundenen Instrumente von "strain-gage-type" fur die Analyse der Gleichgewichtsfunktion des Menschen und Tieres einen guten Erfolg gebracht

ACKNOWLEDGEMENTS

The authors are indebted to Drs G Sakashita, K Terayama H Motohashi, R Koizumi, T Kurosawa, S Kashiwabara, K Sawada and T Sato for their close cooperation in this investigation

The authors wish also to express their deep gratitude to prof M Isaki, Dr H Mitsuoka, Mrs K Mihara and the Rev Javan R Corl for all their kind assistance in composing the English translation of this paper

REFERENCES

DOBIE, W B and ISAAC, P C, 1950 *Electric Resistance Strain Gauges* English Universities Press Ltd

EGMOND A A J, GROEN, J J, and JONGKEES L B W 1952 The function of the vestibular organ *Pract Oto Rhino Laryng 14 Suppl 2*

FUKUDA T, HINOKI M and TOKITA T 1957 Provocation of labyrinthine reflex by visual stimuli *Acta Oto-laryng 48* 425

FUKUDA T, 1957 *Stato-kinetic reflexes in equilibrium and movements* Igaku Shoin Ltd, 268 (in Japanese)

FUKUDA T, 1959 The stepping test Two phases of the labyrinthine reflex *Acta Oto-laryng 50*, 95

FUKUDA T and HINOKI M et al 1961 *The physiology of training* Film presented at the VII International Congress of Oto-Laryng in Paris

HINOKI M and KITAHARA, M, 1961 A study on the physiological relationship between the movements of eyes and head from the view point of the equilibrium function of the human body *Jap Jour Otol Tokyo 64* 604

KITAHARA, M, 1959 Study on the acceleration of the head during human equilibrium movement *The Otol Clinic Kyoto 52* 1044 (in Japanese)

87 Uchimaru Morioka

For example, in a labyrinthine eye nystagmus at first the eye-balls deviate from the normal mid-position relatively slowly but an instant later return much more quickly to the original mid position, thus, a slow and rapid phase in this cycle may be observed to exist These slow and rapid movements are repeated rhythmically to form a labyrinthine nystagmus and it may be stated further that the slow phase is the manifestation of the deviation and the rapid phase, the manifestation of the righting reflex

Therefore, a labyrinthine ataxia should be examined by thinking of it as two separate functions and performing one test for the deviation and another test for that of the righting reflex Thus, we believe that the impairment of the labyrinthine function should be and can be examined more naturally, without the addition of any excessive stimuli and we propose that the following arrangement be adopted

1 Righting reflex
2 Deviation
3 Experimental nystagmus

Through the use of this new arrangement of the vestibular examination it is possible to observe and relate many interesting phenomena, however, since time does not permit me to lecture in great detail on each of the three parts of the new arrangement, we shall confine our lecture to tests related to the righting reflex and deviation with special emphasis on deviation

1 *Tests related to the labyrinthine righting reflex (Balance Test)*

Standing on one foot
Romberg's phenomenon
Von Stein's Goniometer
McNally's Tilting Table

In these tests, a subject after being blindfolded in order to nullify his optic righting reflex, is asked to stand on an unstable base or on a narrow base and then the doctor ascertains whether or not he can maintain the normal position of his head and trunk solely by the use of the labyrinthine righting reflex For example, with McNally's tilting table, the base on which a subject is standing erect or is standing on all fours is inclined by slow degrees in order to observe whether or not the subject can maintain his original position With Romberg's test the feet are brought together pararell to each other in order to reduce the supporting base of the body, also with the test standing on one foot the supporting base is reduced by half Thus, with all these tests the labyrinthine righting reflex, which is used in order to maintain the normal body position may be examined Of course in all these tests, especially in the latter, the deviation also may be observed at the same time by noticing the direction toward which the body tends to fall

2 *Tests related to spontaneous and positional deviation (Imbalance Test)*

Nystagmus
The pointing test
Arm tonus reaction
Fukuda's writing test
Fukuda's stepping test (Unterberger's Tretversuch)

A spontaneous or a positional eye nystagmus is manifest evidence of the imbalance between both labyrinths i e the labyrinthine ataxia In cases where such nystagmus cannot be, or hardly can be observed, tests related to the vestibulo-spinal pathway must be adopted since with such tests the latent imbalance between both labyrinths often can be shown dramatically, especially with our blindfolded vertical writing test as well as with the stepping test, a test which we recently devised from Unterberger's Tretversuch

Fukuda's vertical writing test

The test consists of observing the deviation in a column of letters written vertically with the eyes blindfolded and with the arm kept free from any contact with a desk, etc The pure objectivity of this test is characterized by its results Any deviation in the writing reveal the fact that an imbalance exists between both labyrinthine functions and this imbalance can be evoked by physiological as well as clinical stimuli to the labyrinths that are too weak to induce a nystagmus

For example, during the interval period of Meniere's disease, when the patient wrote his name vertically, Japanese style, with his eyes open, every character showed no deviation, however, when he wrote the same characters blindfolded, every character in each column showed marked deviation every time the test was repeated, even though spontaneous nystagmus as well as past pointing and Romberg's phenomenon could not be observed

Furthermore, this test easily and conveniently may be performed in bed Therefore, the labyrinthine function of a subject even directly after a tympanoplasty may be examined visually as shown in Fig 2, by getting the subject to write his name or any other convenient characters on a sheet of paper We should like to emphasize here, that the patient graphically reproduces his own disease, in this case the latent imbalance between both labyrinths, on paper, not only as a clinical record for the attending physicians but also as a visual indication for the patient himself

The stepping test

This test is a modification of Unterberger's Tretversuch and is superior to the gait test in the examination of the labyrinthine deviation phenomenon as expressed by the lower extremities

A subject with his eyes blindfolded is asked to stand in the center of a circle which has been drawn on the floor and to stretch out both

arms straight before him, then to step by repeatedly raising and lowering first one knee and then the other at a normal walking speed for a total of 50 or 100 steps while attempting to remain at his original starting position

When there is a latent difference between both labyrinthine functions, the subject gradually turns around on his longitudinal axis in a definite direction, although he is unaware of this turning phenomenon

When rotation or displacement of the body occur during the stepping test, this phenomenon is called stepping deviation and its parameters are expressed by the angle of rotation, angle of displacement and distance of displacement as we had shown in a paper (Fukuda, 1959)



Fig 2.

Rather recently, we have gotten a subject to perform the writing as well as the stepping tests while assuming several different positions of the head That is, at first each test is performed with the head in the normal upright position as in the orthodox method Then the same test is repeated first with the head inclined to the right and then to the left, both anteriorly and posteriorly in succession Thus we have been able to show clearly the manifestation of a latent positional imbalance between both labyrinths by the upper as well as the lower extremities even when positional eye nystagmus could not be evidenced

3 *Tests related to an experimental eye nystagmus*
Caloric nystagmus
Postrotatory nystagmus
Cupulometry (The subliminal rotation)

By the above new arrangement of the vestibular examination, it is possible to detect objectively subtle changes in the vestibular function such as its improvement or degeneration during the course of Meniere's disease or the variations of its changes in a patient before and after tympanoplasty

Case Reports
Case No 1

Fig 3 shows the writing of a 22 year old male who was suffering from otitis media purulenta chronica dextra The patient had a 40 to 50 decibel conduction loss of hearing in his right ear, however, he did not complain of any dizziness and neither spontaneous nor positional eye nystagmus could be observed The left column of characters shows the patient's style of writing with his eyes open after he had been admitted to our clinic, whereas the other three columns show his style of writing

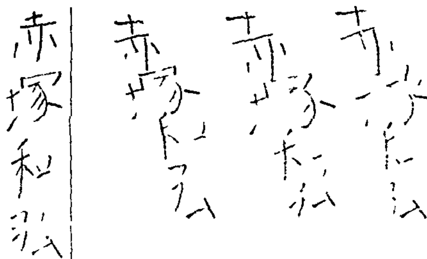


Fig 3.

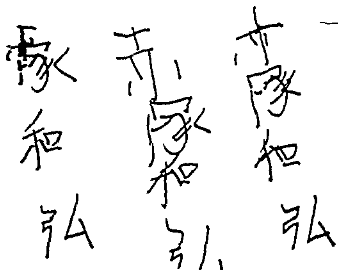


Fig 4

after he had been blindfolded Please notice the marked deviation to the right of 19 to 21 degrees, however, at this time no marked derangement could be detected This clinical manifestation showed an imbalance of both labyrinthine functions that otherwise could not have been detected

Fig 4 shows the style of writing of the same patient the 2nd day after a tympanoplasty had been performed During the patient's operation it was found that his incus and malleus had been destroyed through granulation and that only his stapes remained intact, therefore, a skin graft was performed, with the graft being attached to the stapes After the operation the patient complained of slight dizziness, however, still neither spontaneous nor positional eye nystagmus could be observed Please notice a marked derangement of the characters as well as a marked deviation to the right

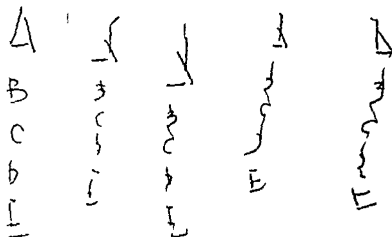


Fig 5

Fig 5 shows the derangement of the letters as well as their marked deviation to the left of another patient written with eyes blindfolded the 1st day after a tympanoplasty, although the left column of letters written with eyes open at the same time shows neither deviation nor derangement This patient also complained of dizziness but as with the preceding patient, *neither spontaneous nor positional eye* nystagmus could be observed

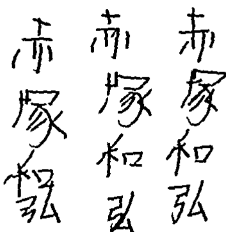


Fig 6

Fig 6 shows the type of writing of the first patient, while being blindfolded the 26th day after his tympanoplasty Please notice that the complete cure of the patient is evidenced by the fact that now his characters are written vertically and without any derangement in all three columns

In addition, the patient's hearing was improved to where he had only a 20 to 30 decibel conduction loss of hearing in his right ear

Case No 2

Fig 7 shows the writing of a 9 year old boy who was suffering from otitis media purulenta chronica sinistra This patient had a 30 to 40 decibel conduction loss of hearing in his left ear, however, as with the first patient he did not complain of any dizziness and neither spontaneous nor positional eye nystagmus could be observed The left column of characters shows the patient's style of writing with his eyes open, after he had been admitted to our clinic, whereas the other three columns show his style of writing after he had been blindfolded Please again notice the deviation, but this time to the right of 10 to 12 degrees and as before no marked derangement could be detected In most cases, the deviation is usually directed toward the ear which is impaired, however, in this case, the deviation was directed toward the unimpaired ear This clinical manifestation showed an imbalance of both labyrinthine functions that otherwise could not have been detected

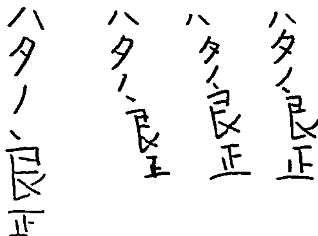


Fig 7

Fig 8 shows the style of writing of the same patient the 1st day after a tympanoplasty had been performed In this case it was found that the incus malleus and stapes were healthy but the middle ear cavity was filled with dirty granulation and puss and this was removed After the operation the patient complained of slight dizziness, however, neither

spontaneous nor positional eye nystagmus could be detected Please notice a marked derangement of the characters as well as a marked deviation of from 20 to 25 degrees to the right

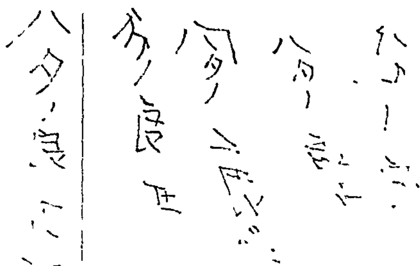


Fig 8

Case No 3

Fig 9 shows the writing of a 28 year old female during an interval between attacks of Meniere's disease This disease was accompanied with tinnitus and the patient had a 20 decibel conduction loss of hearing in her left ear Again neither spontaneous nor positional eye nystagmus could be observed The left column of characters shows the patient's name as written by her in Kanji with her eyes open This column shows

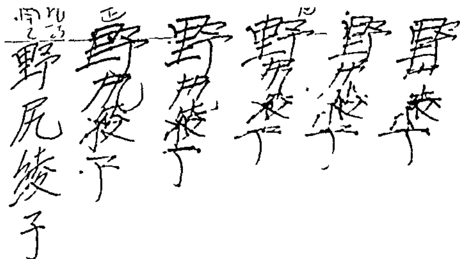


Fig 9



Fig 10

no deviation while the other five columns of the same characters which she wrote with her eyes blindfolded show marked deviation to the left of 18–20 degrees *Again the latent imbalance between both labyrinthine functions clearly is evidenced by this marked deviation*

Fig 10 shows the patient's name in five seperate columns as written by her in Kanji with her eyes blindfolded after she had recieved 20 left stellate blockades one each day for 20 days during the interval between attacks The complete disappearance of the previous deviation evidences and strongly supports the fact that a cure had been effected

Case No 4

Fig 11 also shows the writing of a 25 year old female during an interval between attacks of Meniere's disease As in the previous case

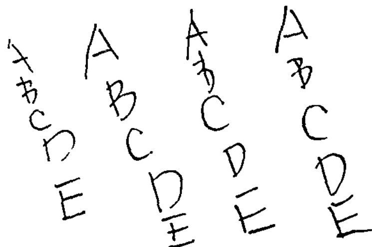


Fig 11

this disease was accompanied with tinnitus but this time the patient ha a 20 decibel conduction loss of hearing in her right ear As before neithe spontaneous nor positional eye nystagmus could be evidenced The fou columns of letters which she wrote with her eyes blindfolded show marke deviation to the right of 20 to 25 degrees, and as before, the latent im balance between both labyrinthine functions may be demonstrated b this deviation

Fig 12 shows six columns of letters The column at the left wa written by the patient with her eyes open after she had received 1 right stellate blockades one each day for 10 days, during the interv between attacks, while the other five columns were written by her afte she had been blindfolded As before, the complete disappearance of th previous deviation evidences and strongly supports the fact that a cur had been effected

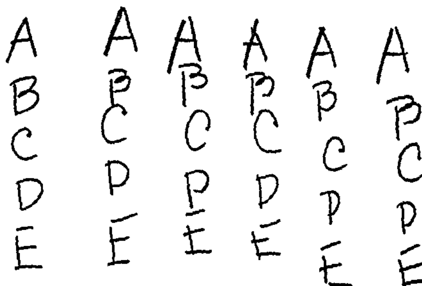


Fig 12

Case No 5

This is the case of a 25 year old male who was suffering from otiti media purulenta chronica dextra A tympanoplasty was performed an the right ear drum was rebuilt with a skin graft from the patient's righ thigh

Fig 13 shows samples of the patient's writing while blindfolded 3 days after the operation In the two columns of letters on the left, deviation to the right of approximately 10 degrees can be observed Thi deviation was the visual proof of an imbalance in the labyrinthine functio which in this case was caused by an obstruction in the Eustachian Tub of the right ear A catheterization was performed, the two columns o letters on the right being samples of the patient's writing immediatel after the Eustachian Tube had been cleared of its obstruction, relievin

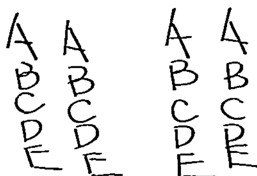


Fig 13.

the imbalance in the labyrinthe functions Thus by the use of the writing test it is possible to detect an imbalance in the labyrinthine functions of even an artificially created ear

Case No 6

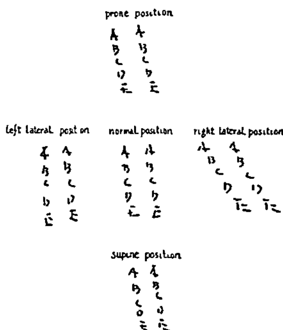


Fig 14.

This is a case of positional vertigo in a 27 year old female with a 20 decibel conduction loss of hearing in the right ear who was suffering from Meniere's disease When the patient's head inclined to the right

slight positional vertigo was induced, whereas manifest spontaneous and positional nystagmus, an objective evidence of vertigo, could not be observed However, while the patient was performing the writing test, an especially marked deviation to the right could be noticed when the head was held in the right lateral position as shown in Fig 14, whereas no deviation could be noticed when the head was held in any of the other positions

Fig 15 shows samples of the patient's writing in all the positions after 10 right stellate blockades had been administered to the patient Please notice that there is only a slight deviation in the writing, when the head was held in the right lateral position After 10 more right stellate blockades the patient was completely cured as could be evidenced by the writing test for after 20 blockades no deviation in any of the head positions could be observed

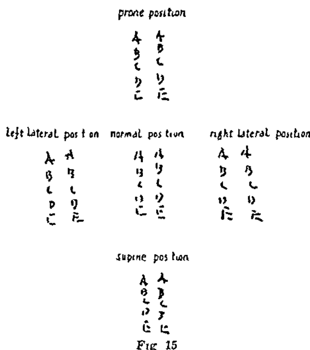


Fig 15

Case No 7

This is another case of positional vertigo, in an 18 year old male with a 25 to 30 decibel conduction loss of hearing in his right ear, who also was suffering from Meniere's disease The patient complained of discomfort in the head and positional vertigo when the head was inclined to the right, although neither spontaneous nor positional nystagmus could be observed However, while the patient was performing the stepping test slight deviation to the right in all head positions could be observed but when the head was inclined to the right, marked deviation with ataxic steps was observed as shown in Fig 16

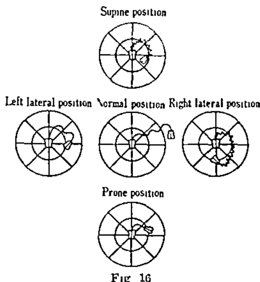


Fig 16

Fig 17 shows the results of the stepping test during treatment by right stellate blockades Please notice the marked deviation with ataxic steps when the head was inclined in the right lateral position A complete cure was effected after 10 right stellate blockades as evidenced by further stepping tests thus this test also provides the doctor with an objective evidence of positional vertigo

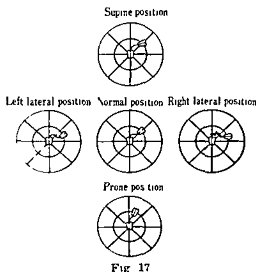


Fig 17

RESUMÉ

L'auteur propose une nouvelle procedure pour l'examen vestibulaire qui consisterait a examiner non seulement le nystagmus experimental, mais aussi le reflexe de redressement ainsi qui le phenomene de la

Notes on Contributors

DR. KENJIRO OWADA is Instructor of Keio University Tokyo. He is interested in patho-physiology of the auditory system and the mechanism of the appearance of nystagmus. Published papers are: The influence of the utricle on nystagmus (Acta oto-laryng 1960 52 215). An interpretation of the appearance of nystagmus in Menière's disease (Acta oto-laryng 1960 52 486). Elektrische Untersuchungen am Nervus ampullaris bei Kaninchen (Arch f Ohren usw Heilk 1960 177 31).

DR. KIYOKO OKUBO is Assistant of Keio University Tokyo.

DR. NAOSHIGE MARUYAMA is Associate Professor of Neurophysiology in the Brain Research Institute of Niigata University. He is chiefly interested in the auditory system and the reflex arc of nystagmus. Most of his work has been done by means of super fine microelectrodes.

DRS. A WATANABE, T KAWASAKI, J KOIZUMI and T HIGUCHI are members of the group of electro-oto-neurology in the Department of Otolaryngology Niigata University.

DR. GENKICHI TOTSUKA is Associate Professor of the University of Tokyo. He was a member of Juntendo University until 1961. He has been investigated the mechanism of cochlear and vestibular reflex. Among his publications are Studies of the Acoustic Reflex (Annals of Otol. etc 63 939 1954). Postrotatory Nystagmus (Acta oto-laryng., 51 570 1959) and A Study of the Vestibulospinal Reflex Electromyographical Observation (Yokohama Med Bull., 11 407 1960).

DR. MUTSURO SUZUKI is a member of otorhinolaryngological clinic of First Tokyo National Hospital.

DR. KISOU KUBOTA is a researcher in Section of Neurophysiology Institute of Brain Research University of Tokyo and now is visiting Department of Physiology University of Oregon. He published a paper titled "Two types of central influence on gamma motor system (J Neurophysiol. 25 309 1962).

DR. SADAO UTSUMI is Professor of Nara Medical College and has been working on the electromyographical studies on the vestibular muscle responses induced by various stimuli upon the labyrinth. His present work is concerned with the vestibular mechanisms in the central nervous system. His published chiefly works: Étude Electromyographique du Reflexe Tonique Labyrinthique (Med Jour Osaka Univer 1957 7 977 960). Étude Electromyographique de la Réponse Musculaire d'Origine Labyrinthique (Med Jour Osaka Univ 195? ? 981 989 and 991 100) and Electromyographical Analysis of the Nystagmus (Jour Nara Med Assoc 1961 12 1090-1095).

DR. HIROSHI SHINNO is Associate Professor of Nara Medical College and his interests are the mechanisms of the posture and the equilibrium of the body in the vestibular system specially in the central nervous system.

DR. TEIZO MUKAI is now the head doctor of the Oto-Rhino-Laryngological Clinic of the Civil Hospital of Yao City in Osaka and had worked in Nara Medical College as Assistant. His major interests is the vestibular reactions following accelerated stimulations. From his principal works: Études des Relations entre la labyrinthe et le Lobule Petrosus Cerebelleux chez le Lapin (Med Jour Osaka Univer 1958 8, 571 578).

DR. YASUTERU YAMANAKA is Instructor of Nara Medical College. From his main published works: Nystagmus (Jour Nara Med Assoc 1962 12 1090-1095).

DR. MASANORI MORIMOTO is Professor of University of Niigata. His chief subject of research has been vestibular physiology and biochemical and electrophysiological investigation of inner ear function. Études biochemiques de l'oreille interne was read at the 58th Congrès Français D'Oto-Rhino-Laryngologie Paris 1960 and Experimental Study on Functional Compensation after the Removal of the Bilateral Auditory Cortexes in Cats at the 5th Interl Congress of Audiology Bonn 1960.

DR. KICHIRO KOIKE is the vestibular research group in Niigata University. His main article was published in Acta Oto-laryngologica 50 4 377 1959 An Observation on the Eye-Speed of Nystagmus".

DRS. K MIZUKOSHI, T OTANI, S IKEDA, Y KATSUMI and T SASAKI are members of the vestibular research group. They have been engaged in study of the physiology of opto-kinetic nystagmus vestibular recruitment and evaluation of the clinical test for equilibrium.

DR. H INO is Director of the E N T department of the Tokyo Second National Hospital. His present mork is connected with the vestibular function in man the vertigo and its diagnosis. Among his publications are: Experimentelle Untersuchungen über das zentral ausgeloste Nystagmusüberwiegen.

DR. HIROSHI SASAKI is professor of Tottori University. His chief research has been concerned with the biochemistry anatomy and physiology of the otolith organ. The most important publications is "Experimentelle Studien über Otolithen (Zschr Laryng 34 740 748 1955)

(Archiv Ohren usw Heilk 179 127 140 1961). Clinical and experimental observations on caloric test (Asian Medical J 13 14 1961). Experimentelle Untersuchungen über Verkürzung der Dauer des Calorischen Nystagmus durch wiederholte thermische Reizungen (Archiv Ohren usw Heilk 179 443-448 1962).

DR. K OKAMOTO is clinical assistant of the E N T department of the Tokyo Second National Hospital. His work has been concerned with the vestibular function and its diagnosis especially the caloric test. He is now studying at the Universitats Hals- Nasen Ohrenklinik München.

DR. TOSHI NAITO is Associate Professor of Osaka University. His major interests have been in the

clinical and pathological problems of Menière's disease and also in the Audiology 1961–1962, he studied some problems of vestibular function in man at Bordeaux University under Professor G Portmann Among the number of his papers "Clinical Studies on Meniere's disease" (Revue de Laryngologie Bordeaux 1962, 83 362) The modification of Portmann's operation for Meniere's disease (Med J Osaka Univ, 1954 5 167)

DR. TOSHIBUMI TATSUMI is Instructor of Osaka University His research has been concerned with physiological acoustics electro-physiology and vestibular function.

DR. TORU MATSUNAGA has been studying in Osaka University He is interested in the central pathway of vestibular system, especially in the function of Reticular Formation of brain stem

DR. TAKASHI MATSUNAGA has been studying in Osaka University His research has been chiefly concerned with the differential diagnosis of disorders complained with Vertigo

DR. ICHIRO KIRIKAE is Professor of the University of Tokyo. His published works includes "Studies of the acoustic reflex (Ann Otol. Rhin. & Laryng 1954 63 939) An experimental study on the fundamental mechanism of bone conduction (Acta Oto-laryng 1959 Suppl. 145 Stockholm) The structure and function of the middle ear" (Monograph, 1960 The University of Tokyo Press) A consideration on the circulation of the perilymph — An experimental study (Ann Otol. Rhin. & Laryng 1961 70 337) "An experimental study on hearing of one's own voice (Pract. Oto-rhino-laryng 1961 23 56) Sudden deafness due to Buerger's disease (Arch. Otolaryng 1962 75 50?) An experimental study of central motor innervation of the laryngeal muscles in the cat (Ann. Otol. Rhin & Laryng 1962 71 222)

DR. JUN ICHI SUZUKI is Assistant in the University of Tokyo. He is interested in the vestibular system and oculomotor mechanisms At present he is studying with Dr Morris B Bender in the Department of Neurology The Mount Sinai Hospital, New York Among his publications are "Post rotatory nystagmus Modifications observed in experiments with repeated rotatory stimulation (Acta Oto-laryng 1959 51 5 0) A study on pendular nystagmus Its contribution to the understanding of nystagmus mechanisms (Acta Oto-laryng 1961 53 361) "Clinical application of optokinetic nystagmus" (Acta Oto-laryng 1962 54 49)

DR. KOJI TOKUMASU is Assistant of the University of Tokyo His main interests are in labyrinthine physiology

DR. YUTAKA TSUIKI was Professor of Tohoku University from 1941 to 1962 His doctorate dissertation was an "Experimental study on the central mechanism of vestibular nystagmus published in Vol. 24 (1931) of Fukuoka Acta Medica. Japan Among his numerous research works, the study on ménière's disease may be mentioned as most outstanding

DR. SHUICHI KATAGIRI is Professor of Tohoku University His doctorate dissertation was entitled "Experimental Studies of the Influence of Rotation on the Ear published in Vol. 54 (1952) of Jap Jour Otol., Tokyo. His principal research works are i) studies on malignant tumor of nasal and paranasal cavity and ii) studies on local anaesthesia in otorhinolaryngology

DR. JIRO HOZAWA is Instructor of Tohoku University He was awarded on he has been engaged chiefly in i) the study on cupulometry and ii) the clinical study on Meniere's disease

DR. MIYUKI KAKUTA is at present active in the Hanamaki Hospital.

DR. MASAMI USAMI is an Assistant of Tohoku University

MISS KAZUKO SATO is now in Tohoku University in the capacity of loctor course student. She has been chiefly engaged in the study on various factors influencing experimental nystagmus

DR. TOSHIO SHIRAIWA is Professor of Tokyo Medical College His investigations have been in the field of phonology and vascular physiopathology of the nose with particular emphasis upon the phenomena of hemorrhage Lately he has extended his studies to the labyrinthine function and the hearing tests of children.

DR. MANABI HINOKI is Professor of Iwate Medical College His research has been chiefly concerned with the study of the physiology of training formation of the equilibrial reflexes through the daily repetition of visual or labyrinthine stimulation His main interests also lie in the field of analysis and treatment of vertigo due to the oto-neurological diseases In addition to the investigations above mentioned his present works concerns with the following problems Physiological relationship between the ocular and the spinal reactions in case of active body movements in human subjects and animals

DR. MASAAKI KITAHARA is Associate Professor of Iwate Medical Collage His work concerns the investigations of equilibrium function in human subjects

DR. T FUKUDA is Professor of the Gifu Medical School. His main interests lie in the field of physiology of vestibular apparat, with particular emphasis upon the new tests of vestibulo-spinal reaction and functional development of labyrinthine function with training His published work is "Studies on Human Dynamic Postures from the View Point of Postural Reflexes" (Acta oto-laryng supp 161 1961)

DR. T TOKITA is Associate Professor of the Gifu Medical School.

*

COVER

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta
OTO-LARYNGOLOGICA

S U P P L E M E N T U M 180

THE USE OF MASKING NOISE AND ITS LIMITATION IN CLINICAL AUDIOMETRY

BY

E. KÖNIG

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM O

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 180

THE USE OF MASKING NOISE
AND ITS LIMITATION IN CLINICAL AUDIOMETRY

By E Konig

Basel, Switzerland

From the Electro-acoustic Laboratory (Head E Konig)
University Clinic of Oto-Rhino-Laryngology
(Director Prof E Luscher M D) Basel

INTRODUCTION

Among the serious difficulties which arise in clinical audiometry is the exclusion of the untested ear by means of a masking sound The principle of masking is to lead to the non tested cochlea a sound capable of covering efficiently the air or bone conducted stimulus originating in the tested ear, without overmasking, or raising of the hearing threshold level (for the test signal) of the ear under test Although masking may theoretically be accomplished by almost any sound (pure tones, noise produced by a jet of water or air impinging on the eardrum, electronically generated broad band of noise), it is generally accepted [5, 23 26, 34, 36, 50, 57, 60, 61, 65 82, 83, 90–92, 96, 97, 108, 122, 123, 136, 148] that a narrow band of noise centered on the test tone is the most suitable sound for masking purposes The principal advantage of narrow band over broad band noise lies undoubtedly on the fact, that the former may be accurately adjusted to the ear's frequency response, furthermore, according to the various studies of the critical masking band [16, 27, 31, 32, 45, 54, 80, 119, 146] a narrow band of noise matched to the frequency of the test tone permits masking of this pure tone with a minimum of sound energy

On the assumption that the relation between the amount of masking and the sound pressure level of the masking stimulus is linear (i e , each additional decibel of noise produces an additional decibel of masking), and taking into account the various factors which may play an important role in air and bone conduction tests, several authors [5, 37, 83, 91, 92, 99 107, 121, 124, 128] have already pointed out in the last decade how we can calculate the limits within which the intensity of the masking noise must be maintained in order to eliminate efficiently the test tone in the untested ear without fear of cross masking effects However, because of certain discrepancies which exist in the results of these analyses, we shall consider in the present report the various possible pathways of the sound radiated by the transducers and discuss the limitations of the most popular methods actually used in routine audiometry

CHAPTER I

MEASUREMENT OF AIR CONDUCTION

A TRANSMISSION PATHWAYS

Fig 1 shows the arrangement of the earphones which is commonly employed for air conduction tests The various lines and arrows represent schematically the different possible pathways of the test signal (S) and the masking noise (N) which are emitted by the receivers R_1 and R_2 respectively, whereas the corresponding transmission losses are indicated by different symbols An explanation of all the symbols used in this report is presented in the appendix of this paper

Normally, the sound radiated by an earphone (at threshold intensity) reaches the labyrinth of the ipsilateral ear by air conduction (i e , through the oval window by way of the auditory canal and the middle ear) It is however possible that, in the case of a severe middle ear deafness the sound generated by an air conduction receiver (at threshold intensity) elicites a sensation of hearing in the ipsilateral ear by bone conduction (transtemporal pathway) Accordingly, if the transmission losses of the test signal in passing through the middle ear to the cochlea of the ear under examination (transtympanic transmission losses) are higher than the transtemporal transmission losses one is unable to determine the true air conduction hearing threshold level of the ear under test In such cases the false audiometrically measured air-conduction hearing level for the test signal (provided the untested ear may be blocked without cross masking effects) is equal to the amount of the transtemporal transmission loss $TL_{BSR(1)}$ plus the bone conduction hearing threshold level of the ear under test $HL_{BS(1)}$ Therefore in clinical audiometry the larger measurable middle ear transmission loss (bone air gap ') for a given test signal is limited by the amount of the transtemporal insulation

As it is already well known [4 5, 10 13, 17–19, 23 34, 36 37 41, 49–51, 56–58, 64 66 68 72–76 81–83 87 91–94, 98 101, 104 105 117 118 121, 124, 128, 129, 131 137 142 143 149] when the hearing of the ear being tested is poorer than the untested ear, the test signal may be transmitted to the cochlea of the untested ear (shadow hearing effect) through the skull (transcranial pathway) or into the external auditory canal of the untested ear through the pericranial pathway (air and/or metal headband) Reciprocally at higher intensities, the masking noise can also stimulate the ear under test in passing either through the skull or the air (cross masking effect) In this connection it should however be noted that the acoustical leakage between the two ears (as also the transtemporal leakage) arises usually from the vibrations imparted to the skull at the air bone interface bounding the receiver According to Littler Knight and Strange [87] and Zwislocki [149] the amount of the transcranial transmission loss increases as the area of the head exposed to the air conducted sound

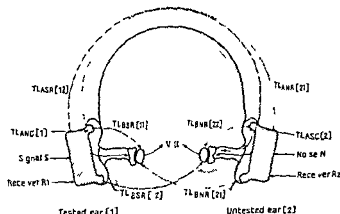


Fig. 1. Schematic sketch showing the arrangement of the receivers R_1 and R_2 for measurement of the air conduction hearing (threshold) level. The various possible pathways of the test signal and the masking noise (generated by the receiver R_1 and R_2 respectively) are represented by different lines and arrows.

waves decreases. Thus, when ordinary air conduction receivers of commercial audiometers are used the amount of the transcranial attenuation usually attains values as low as 45–60 db [74, 82, 91, 92, 98–101, 105, 128, 149], whereas by introducing the sound into the ear through a perforated rubber ear plug the interaural attenuation may increase to figures as high as 70–90 db [57, 105, 149].

In the above description of the various sound pathways it was presumed that a receiver was applied only to one ear. If each ear is covered with an earphone two additional factors must be taken into consideration, i.e. the transmission losses through the cushions of the receivers and the bone conduction threshold shift due to the occlusion of the auditory canal [2, 3–7, 9, 30, 33, 35, 40, 42–49, 59, 63, 84, 88, 99, 100, 103–105, 109–111, 125, 132, 140, 141, 144]. The first factor contributes to increase the total amount of the pericranial transmission losses, whereas the second one may in certain cases reduce the effective amount of the transcranial attenuation. Normally, for frequencies up to 1000 c.p.s. the occlusion of the meatal opening causes a bone conduction threshold gain of 10–15 db, at the higher frequencies this effect decreases while above 2000 c.p.s. no threshold shift occurs [59, 103]. It is important here to emphasize that the occlusion effect is also normally present in the case of a pure cochleo-neural deafness, whereas, in the case of a middle ear deafness, the magnitude of this effect depends on the amount of the transtympanic transmission loss [59, 103]. Moreover, it is interesting to note that Huizing [59], who has registered with the flat type earphones of the Peters, Peekel and Atlas audiometer bone conduction threshold shifts of 5–10 db for the low frequencies, found no such occlusion effect when the loudspeaker type of the Danish Pedersen audiometer described by Groen [45] was used.

B. AUDITORY MASKING AND THE NARROW BAND OF NOISE

Before we analyse some particular hypothetical cases, it seems worthwhile to recall here briefly the general rules governing the practical application of masking in audio-

metry and the different types of narrow band of noise generally recommended in the literature

Although auditory masking arises mainly from the cochlear activity pattern of the masking sound [28, 29, 126] the presence of a masking noise in the untested ear elevates slightly the auditory threshold of the ear under test even if that latter ear does not receive the masker [23, 83, 92, 99, 104, 149] This interaural interference component (central masking), which must not be confused with the normal clinical masking, varies from one person to another, the tested ear s threshold is usually raised to a level as high as 5 to 15 db [83] According to Liden, Nilsson and Anderson [83] central masking is probably mediated by efferent pathways [43, 114] Since the effect of central masking is usually somewhat reduced when the masking sound is continuous and the test tone intermittent [92] other additional factors, such as fatigue [96] and listener distraction [133] may also play a certain part

Despite the inconveniences outlined above, the application of masking in conventional audiometry is not too restricted, if the masking noise fulfills the following requirements

a) The intensity of the masking noise in the untested ear must be raised at least to the level at which the test tone in the untested ear is effectively masked In the following we shall call this limit the ' minimum effective intensity ' of masking noise $Imin_{N(2)}$ *(expressed in decibels above the normal threshold of hearing for that noise)*

b) The sound pressure level of the masking noise must not surpass the level ("maximum allowable intensity ' of masking noise $Imax_{N(2)}$, measured in decibels above the normal threshold level of that noise) at which masking of the test signal at the ear under examination begins (cross masking effect, which must not be confused with central masking)

c) The sound pressure level of the masking noise must not exceed the patient's discomfort level [5, 83] of the untested ear $D_{N(2)}$ (expressed in decibels above the normal threshold of hearing for that noise)

d) Since masking is always a tiring and irritating procedure for the patient even when his discomfort threshold is not approached [83], the loudness of the masking sound has to be maintained as low as possible

From the first three above requirements (a, b and c) it appears clearly that the untested ear can be successfully blocked whenever the minimum effective intensity of masking noise is lower than the maximum allowable intensity of masking noise and the patient's discomfort level of the untested ear In regard to the last requirement (d) a narrow band of noise whose chief components are centered around the test tone is undoubtedly the most appropriate masking sound for clinical purposes As it is well known [57 82, 148], in contrast to the masking effect of a wide band noise the masking efficiency of a narrow band (i e , the relationship of masking level or masking ability to loudness) is relatively higher Moreover, the narrow band noise has practically the same threshold as the test tone and can be calibrated similarly for each frequency individually [148]

Whereas the narrow bands of noise generally recommended by a large number of

authors [26, 57, 60, 61, 82, 96, 97] are obtained with the aid of a so called white noise generator (compensated continous spectrum) and a set of selectable band pass filters, the narrow band noise, described by Zwislocki [148] as early as 1951, is generated by means of a modulator with carrier suppression. In this last method, the carrier (test tone) is modulated in the modulator by a distorted 50 cycle voltage derived from the mains. Owing to the symmetrical layout of the system the carrier frequency (test tone) is cancelled out and practically only six discrete frequencies come out of the modulator, which lie at 50, 100 and 150 c.p.s. above and below the carrier frequency, with amplitude approximately in the ratio 25:5:1. This noise has practically the same effect as a narrow band noise with a compensated continous spectrum centered around the test tone. Therefore similar kinds of narrow band noise signals which consist of a group of relatively closely adjacent discrete tones (discontinous spectrum) may also be applied for clinical purposes. Such mixtures of constant discrete tones, however, should not, if possible, contain the frequency of the test tone, in order to obviate the risk of a binaural summation [148]. In this connection, it is particularly interesting to note that Greenwood [45] used recently, for his investigations on the critical band, narrow band noises which were produced by modulating in a similar manner as in the technique applied by Zwislocki, the carrier in the ring modulator by a low pass filtered white noise.

In order to be able to evaluate the minimum effective intensity of masking noise, an important question to ask is the extent to which the pure tone masking produced by narrow bands of noise depends on the type or nature of the hearing impairment. According to Jerger, Tillman and Peterson [61], who have measured the masking of sinusoids by three octave bands of thermal noise in normal and hearing impaired ears, the difference of the results obtained by different groups of deafness is very little for the frequencies within the noise band. Therefore, if we know the accurate value of the just necessary increment above its threshold level of the intensity of a specified narrow band noise (expressed in decibels) for masking the test signal (at threshold intensity) in a normal ear $[\Delta I^*_{N(1)}]$, it is possible on the basis of the other different quantities involved to determine with a certain margin of safety the intensity of noise, which apparently will be sufficient to block the untested ear, without overmasking or elevating the auditory threshold of the ear under examination.

C. PARTICULAR HYPOTHETICAL CASES

1. Cochleo-neural deafness

Seeing that in the cases of pure cochleo neural deafness the problem of masking is not difficult we shall at first analyse the hypothetical case of a unilateral cochleo-neural deafness. In the following examples as well as in the different discussions of this paper, it will always be assumed that the bone conduction of the ear under test is poorer than the bone conduction of the untested ear, furthermore, we shall suppose that the minimum effective intensity of masking noise does not attain a level at

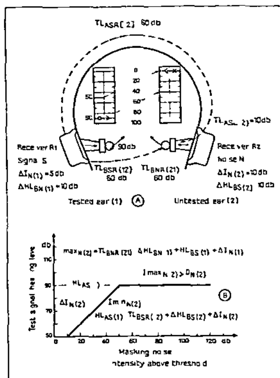


Fig 2 A Schematic representation of the various important pathways of the test signal and

levels for the test signal (expressed in decibels relative to the zero reference level of the audiometer) of the ear under test and the untested ear are indicated on the small graphs by the following symbols

—o— Air conduction hearing (threshold) level for the test signal of the ear under test [$HL_{AS(1)}$]

> Bone conduction hearing (threshold) level for the test signal of the ear under test [$HL_{BS(1)}$]

—x— Air conduction hearing (threshold) level for the test signal of the untested ear [$HL_{AS(2)}$]

< Bone conduction hearing (threshold) level for the test signal of the untested ear [$HL_{BS(2)}$]

Fig 2 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity (expressed in decibels above the normal threshold of hearing for that noise) In Figs 2 B to 6 B 8 B to 10 B, 12 B to 14 B, 16 B to 19 B and 21 B to 24 B the ordinates of the graphs show the intensity levels (measured in decibels relative to the zero reference level of the audiometer) at which the test signal is just perceived in the untested ear or in the ear under test when the intensity of the masking noise is gradually increased

which the patient s responses may be heavily affected either by discomfort or central masking Depending on whether the effective amount of the transcranial transmission losses of the test signal [$TL_{BSR(12)} - \Delta HL_{BS(2)}$] is lower or higher than the corresponding total amount of the pericranial transmission losses [$TL_{ASR(12)} + TL_{ASC(2)}$], we must distinguish the two following cases

a) $TL_{ASR(12)} + TL_{ASC(2)} > TL_{BSR(12)} - \Delta HL_{BS(2)}$

Figs 2 A and B illustrate schematically the problems which must be confronted, *when one is determining in cases of pure unilateral cochleo neural deafness the air-*conduction hearing threshold level of the impaired ear (right) The hypothetical air and bone conduction hearing threshold levels for the test signal of the ear under examination and the untested ear (expressed in decibels relative to the zeroreference level of the audiometer) are indicated on the small graphs of Fig 2A by different symbols, moreover the various lines and arrows of this figure represent schematically the most important possible pathways of the test signal and the masking noise

Taking into account the different quantities specified in Fig 2A, the test signal generated by the receiver R_1 is just perceived in the untested ear by bone conduction (transcranial shadow hearing) when its intensity is raised to a level as high as 50 db [$TL_{BSR(12)} - \Delta HL_{BS(2)}$] above the normal threshold of hearing for that signal This fact is indicated in Fig 2A by the oblique (dash dot) upper line drawn between the two small graphs Thus according to the hypothetical data of Fig 2A the intensity of the masking noise which is lead to the untested ear, must be adjusted to a level of at least 10 db [$\Delta I_{N(2)}$] above threshold in order to mask effectively the test signal in the ear not being examined (normal ear) On the assumption that the relationship between the amount of masking (threshold shift for the test signal) and the masking noise level (expressed in decibels above the normal threshold of hearing for that noise) is linear one can study as it is schematically shown in Fig 2B, how the hearing threshold level for the test signal varies when the masking noise level is increased step by step The graph of Fig 2B clearly demonstrates the fact that, from a test signal level of 90 db any further increment of the masking noise intensity does not produce an additional threshold shift for the test signal, there is a plateau The level *of this plateau corresponds to the level [$HL_{AS(1)}$] at which the test signal begins to* evoke an auditory sensation in the ear under test At a test signal level of 90 db and in the absence of masking noise the test signal attains in the ear not under test a sensation level of 40 db [$HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)}$] above threshold The minimum effective sound pressure level of the masking noise just necessary to block the untested ear is thus equal to 50 db [40 db + $\Delta I_{N(2)}$] above the threshold of hearing

Accordingly in the cases of pure cochleo neural deafness where for a given test signal the shadow hearing effect occurs by bone conduction the minimum effective intensity of masking noise may be derived from the following formula

(1a) $I_{min\,N} = HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)}$

In a similar manner one can also easily calculate the maximum allowable intensity *of masking noise If one supposes that in the example outlined in Figs 2A and B* the ear under test is normal the masking noise would begin to affect the threshold of audibility for the test signal of the ear under test (transcranial cross masking) as soon as the masking noise delivered to the untested ear reaches 55 db [$TL_{BNR(21)} - \Delta HL_{BN(1)} + \Delta I_{N(1)}$] above the threshold of hearing for that noise Seeing that in the hypothetical case of Fig 2A the bone conduction hearing threshold level for the test signal of the ear under test is equal to 90 db [$HL_{BS(1)}$] the maximum allowable inten-

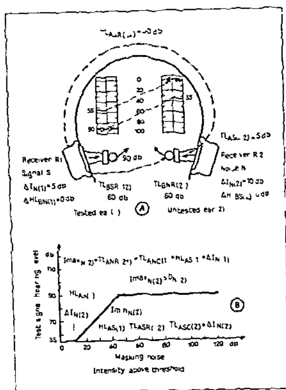


Fig 3A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receivers R_1 and R_2 respectively) in the hypothetical case of a unilateral cochleo neural deafness, and where it is further supposed that $TL_{ASR(12)} + TL_{ASC(2)} < TL_{BSR(12)} - \Delta HL_{BS(2)}$

Fig 3B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

sity of masking noise reaches thus a level as high as 145 db (55 + 90 db) above the threshold of hearing

The foregoing considerations lead one to the general conclusion that, whenever cross masking effects occur chiefly by bone conduction (transcranial cross masking), the maximum allowable intensity of masking noise is given by

$$(2a) \qquad Imax_{N(2)} = TL_{BNR(21)} - \Delta HL_{BN(1)} + \Delta I_{N(1)} + HL_{BS(1)}$$

$$b) \quad TL_{ASR(12)} + TL_{ASC(2)} < TL_{BSR(12)} - \Delta HL_{BS(2)}$$

In contrast to the preceding example, Fig 3A presents an hypothetical case where it is supposed that air conduction (pericranial pathway) is chiefly responsible for the acoustical leakage between the ears According to the data of Fig 3A, in the absence of a masking noise the test signal applied to the ear under examination (impaired ear) just elicites a sensation of hearing in the untested normal ear when its intensity is adjusted to a level equal to 55 db [$TL_{ASR(12)} + TL_{ASC(2)}$] above the normal threshold of hearing At a test signal level of 90 db (air conduction hearing threshold level for the test signal of the ear under examination) the sensation level of the test signal

in the untested ear (without masking) is then as high as 35 db [$HL_{AS(1)} - TL_{ASR(12)} - TL_{ASC(2)}$] Thus, the minimum effective sound pressure level of the masking noise is equal to 45 db [$35 + \Delta I_{N(2)}$] above the normal threshold of hearing Finally, under the above hypothesis, one obtains then the following formula

(1 b) $\quad Imin_{N(2)} = HL_{AS(1)} - TL_{ASR(12)} - TL_{ASC(2)} + \Delta I_{N(2)}$

On the assumption that cross masking effects may also occur through the pericranial pathway, the masking noise which is lead to the untested ear would evoke a sensation of hearing in a controlateral normal ear, when the level of that noise (expressed in decibels above the normal threshold of hearing) is equal to $TL_{ANR(21)} + TL_{ANC(1)}$ Therefore, if we suppose that, as in the case of Fig 3A, the tested ear has an abnormal air conduction hearing threshold level, one can derive the maximum allowable intensity of masking noise from the expression

(2 b) $\quad Imax_{N(2)} - TL_{ANR(21)} + TL_{ANC(1)} + HL_{AS(1)} + \Delta I_{N(1)}$

On the basis of the expressions (1a, 1b, 2a and 2b), it follows that in the cases of pure cochleo neural deafness the air conduction test for a given pure tone may be carried out without fear of cross masking effects whenever the following conditions are satisfied

(3 a) $\quad HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} < TL_{BNR(21)} - \Delta HL_{BN(1)} + \Delta I_{N(1)} + HL_{BS(1)}$
and

(3 b) $\quad HL_{AS(1)} - TL_{ASR(12)} - TL_{ASC(2)} + \Delta I_{N(2)} < TL_{ANR(21)} + TL_{ANC(1)} + HL_{AS(1)} + \Delta I_{N(1)}$

Usually one can assume that for narrow band noises $TL_{BSR(12)} \cong TL_{BNR(21)}$, $TL_{ASR(12)} \simeq TL_{ANR(21)}$, $TL_{ASC(2)} \simeq TL_{ANC(1)}$ and $\Delta I_{N(2)} \simeq \Delta I_{N(1)}$ Furthermore seeing that in cases of pure cochleo neural deafness $\Delta HL_{BS(2)} \cong \Delta HL_{BN(1)}$ and $HL_{AS(1)} = HL_{BS(1)}$ one obtains finally

(4 a) $\quad TL_{BSR(12)} - \Delta HL_{BS(2)} > 0$ and

(4 b) $\quad TL_{ASR(12)} + TL_{ASC(2)} > 0$

From the expressions (4a and 4b) it appears clearly that, as expected, the exclusion of the untested ear by means of a masking noise presents no serious difficulties in the cases of pure cochleo neural deafness

2 *Mixed deafness*

In the cases of mixed deafness the margin of safety with which the unwanted ear can be blocked depends on the degree of the middle ear impairments In the following we shall examine four hypothetical cases of mixed deafness which are particularly interesting from the audiometrical point of view

a) $HL_{AS(1)} - HL_{BS(1)} \leq TL_{BSR(11)}$ and $HL_{AN(2)} - HL_{BN(2)} \leq TL_{BNR(22)}$

Figs 4 A and B show schematically the various important factors involved when one is performing air conduction measurements in cases of mixed deafness where the

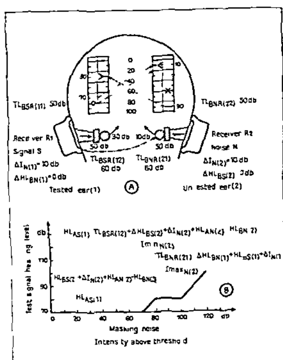


Fig 4A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receivers R_1 and R_2 respectively) in the hypothetical case of a mixed deafness, and where it is further supposed that $HL_{AS(1)} - HL_{BS(1)} = TL_{BSR(11)}$ and $HL_{AN(2)} - HL_{BN(2)} = TL_{BNR(22)}$

Fig 4B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

amounts of the transtympanic transmission losses are equal to or lower than those of the transtemporal transmission losses As one can deduce from the graphs of Figs 4A and B the minimum effective intensity of masking noise is equal to the masking noise intensity which is just necessary for excluding effectively in the cases of pure cochleo neural deafness the test signal in the untested ear $[HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)}]$ plus the amount of the transtympanic transmission loss of the ear not being tested $[HL_{AN(2)} - HL_{BN(2)}]$ Thus the minimum effective intensity of masking noise is given by

(5a) $$I_{min\,N(2)} = HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)}$$

On the assumption that the total amount of the pericranial transmission losses of the masking noise applied to the untested ear is higher than the corresponding effective amount of the transcranial transmission losses, the maximum allowable intensity of masking noise can be calculated from the formula (2a) Thus, under the above hypothesis it is possible to perform correctly air conduction tests, whenever the following requirement is fulfilled

(6a) $$HL_{AS(1)} - TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(21)} - \Delta HL_{BN(1)} + HL_{BS(1)} + \Delta I_{N(1)}$$

in the untested ear (without masking) is then as high as 35 db [$HL_{AS(1)} - TL_{ASR(12)} - TL_{ASC(2)}$] Thus, the minimum effective sound pressure level of the masking noise is equal to 45 db [$35 + \Delta I_{N(2)}$] above the normal threshold of hearing Finally, under the above hypothesis one obtains then the following formula

(1 b) $\quad Imin_{N(2)} = HL_{AS(1)} - TL_{ASR(12)} - TL_{ASC(2)} + \Delta I_{N(2)}$

On the assumption that cross masking effects may also occur through the pericranial pathway, the masking noise which is lead to the untested ear would evoke a sensation of hearing in a controlateral normal ear when the level of that noise (expressed in decibels above the normal threshold of hearing) is equal to $TL_{ANR(21)} + TL_{ANC(1)}$ Therefore if we suppose that, as in the case of Fig 3A, the tested ear has an abnormal air conduction hearing threshold level one can derive the maximum allowable intensity of masking noise from the expression

(2b) $\quad Imax_{N(2)} \quad TL_{ANR(21)} + TL_{ANC(1)} + HL_{AS(1)} + \Delta I_{N(1)}$

On the basis of the expressions (1a 1b 2a and 2b), it follows that in the cases of pure cochleo neural deafness the air conduction test for a given pure tone may be carried out without fear of cross masking effects whenever the following conditions are satisfied

(3a) $\quad HL_{AS(1)} \quad TL_{BSR(12)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} < TL_{BNR(21)} \quad \Delta HL_{BN(1)} + \Delta I_{N(1)} + HL_{BS(1)}$

and

(3b) $\quad HL_{AS(1)} \quad TL_{ASR(12)} \quad TL_{ASC(2)} + \Delta I_{N(2)} < TL_{ANR(21)} + TL_{ANC(1)} + HL_{AS(1)} + \Delta I_{N(1)}$

Usually one can assume that for narrow band noises $TL_{BSR(12)} \cong TL_{BNR(21)}$, $TL_{ASR(12)} \simeq TL_{ANR(21)}$, $TL_{ASC(2)} \simeq TL_{ANC(1)}$ and $\Delta I_{N(2)} \simeq \Delta I_{N(1)}$ Furthermore seeing that in cases of pure cochleo neural deafness $\Delta HL_{BS(2)} \cong \Delta HL_{BN(1)}$ and $HL_{AS(1)} = HL_{BS(1)}$ one obtains finally

(4a) $\quad TL_{BSR(12)} \quad \Delta HL_{BS(2)} > 0$ and

(4b) $\quad TL_{ASR(12)} + TL_{ASC(2)} > 0$

From the expressions (4a and 4b) it appears clearly that as expected the exclusion of the untested ear by means of a masking noise presents no serious difficulties in the cases of pure cochleo-neural deafness

2 *Mixed deafness*

In the cases of mixed deafness the margin of safety with which the unwanted ear can be blocked depends on the degree of the middle ear impairments In the following we shall examine four hypothetical cases of mixed deafness which are particularly interesting from the audiometrical point of view

a) $HL_{AS(1)} \quad HL_{BS(1)} \leq TL_{BSR(11)}$ and $HL_{AN(2)} \quad HL_{BN(2)} \leq TL_{BNR(22)}$

Figs 4 A and B show schematically the various important factors involved when one is performing air conduction measurements in cases of mixed deafness where the

$TL_{BSR(11)} = TL_{BSR(22)} = TL_{BSR(12)}$ furthermore $HL_{AS(1)} - HL_{BS(1)} = TL_{BSR(11)}$ and $HL_{AS(2)} - HL_{BS(2)} = TL_{BSR(22)}$ it is impossible (in accordance with the expression 7a) to find a plateau when one increases the masking noise intensity step by step In such a case, whatever the conventional method used, we are not able to determine correctly the air-conduction hearing threshold level of the ear with the poorer bone conduction

b) $HL_{AS(1)} - HL_{BS(1)} < TL_{BSR(11)}$ and $HL_{AN(2)} - HL_{BN(2)} > TL_{BNR(22)}$

Figs 5A and B illustrate a hypothetical case where the amounts of the transtympanic transmission losses of the ear under test and the untested ear are respectively lower and higher than the corresponding amounts of the transtemporal transmission losses Referring to the graph of Fig 5B, it follows that one can block the untested ear without the risk of cross masking whenever the following conditions are satisfied

(6b) $$HL_{AS(1)} - TL_{BSR(12)} + \Delta I_{N(2)} + TL_{BNR(22)} < TL_{BNR(21)} - \Delta HL_{BN(1)} + HL_{BS(1)} + \Delta I_{N(1)}$$

For simplicity let us assume that $TL_{BNR(21)} = TL_{BSR(21)} = TL_{BNR(22)} = TL_{BSR(22)}$, $\Delta I_{N(2)} \cong \Delta I_{N(1)}$ and $\Delta HL_{BN(1)} \cong 0$ (higher frequencies or severe middle ear impairment)

One obtains then

(7b) $$HL_{AS(1)} - HL_{BS(1)} < TL_{BSR(12)} = TL_{BSR(11)}$$

Accordingly, although it is impossible under the above hypothesis to determine the true air conduction hearing threshold level of the ear which suffers from a severe middle ear impairment (because of the unavoidable transtemporal pathway), one can carry out the air conduction measurement for the ear having the smaller middle ear disturbance with a certain margin of safety (provided the level of the minimum effective intensity of masking noise does not exceed the maximum intensity limit of the audiometer)

c) $HL_{AS(1)} - HL_{BS(1)} > TL_{BSR(11)}$ and $HL_{AN(2)} - HL_{BN(2)} > TL_{BNR(22)}$

Figs 6A and B allow us to analyse a particular case where it is supposed that the amount of the transtympanic transmission losses of each ear exceeds the amount of the transtemporal transmission losses Although, as we already know, it is impossible in such cases to determine the true air conduction hearing threshold levels of any ear one must nevertheless be able to predict the conditions under which there may be the danger of cross masking effects Referring to the graph of Fig 6B, cross masking effects can be avoided, if the following requirement is fulfilled

(6c) $$TL_{BSR(11)} + HL_{BS(1)} - TL_{BSR(12)} + TL_{BNR(22)} + \Delta I_{N(2)} < TL_{BNR(21)} + HL_{BS(1)} + \Delta I_{N(1)}$$

Supposing that $TL_{BSR(11)} = TL_{BNR(22)}$, $TL_{BSR(12)} = TL_{BNR(21)}$, $\Delta I_{N(2)} = \Delta I_{N(1)}$ one obtains then

(7c) $$2(TL_{BSR(12)} \quad TL_{BSR(11)}) > 0$$

Thus under the above assumptions the margin of safety with which the test signal can be eliminated from the untested ear without elevating the auditory threshold of

CHAPTER II

GENERAL REMARKS CONCERNING THE MEASUREMENT OF BONE CONDUCTION

Among the important factors which must be taken into consideration when one performs bone conduction tests are the effects due to the choice of the application point of the bone conduction vibrator [86] and the static coupling force with which the vibrator is maintained against the head [2 8 9, 44, 52, 69 70 84] For many years the mastoid process and the frontal bone site have been used in clinical audiometry for the determination of the bone conduction hearing threshold level The reason why the mastoid process has long been preferred by a larger number of investigators was perhaps based on the false opinion that the ear under examination could be tested more or less independently of the untested ear However it has been emphasized repeatedly in the literature that placing the vibrator on the forehead is much more satisfactory than applying the vibrator to the mastoid process [6, 7, 12 53, 56 60 69 70, 85 86 91 92 99 100] The principal advantages of the frontal bone site over the mastoid process may be summarized as follows

a) It is easier to fasten the bone conduction vibrator to the forehead by means of a headband than it is to apply the vibrator to the mastoid process Thus seeing that the bone conduction hearing threshold level for a given frequency depends on the pressure exerted on the vibrator [69 70] the errors in the determination of the bone conduction curve may be somewhat reduced when the vibrator is placed on the fore head

b) At the frontal bone the tissues are more homogeneous over a reasonable area [56] A displacement of the bone conduction vibrator away from the middle of the forehead gives rise to only relatively small fluctuations in the bone conduction [6 7 12, 67], whereas variations of only a few millimeters in the position of the vibrator along the mastoid process may cause sensitivity changes of 10 db or more [7 12] Thus when the frontal bone site is used for bone conduction tests it is not necessary (as with the mastoid) to search for the most sensitive point

c) The risk of the sound radiated by the vibrator reaching the inner ear by air conduction is lower

d) With bone conduction through the forehead there is no necessity to change the position of the vibrator when testing both the patient s ears

e) When the vibrator is applied to the mastoid the oscillations of the soft parts of the acoustic duct will probably set the auditory ossicles in motion so that the tone is not heard purely by bone conduction but also through the auditory ossicles [6]

f) On the forehead the tactile sensations that arise at the low frequencies are more easily discriminated from the sounds [12]

g) Bone conduction depends, among many other factors, also on the relative motion of the auditory ossicles with respect to the tympanic cavity [6, 7, 11, 67, 79, 127, 144]. The theoretical work of Barany [6] and the investigations of Link and Zwislocki [86] and Kirikae [67], however, show that bone-conduction testing by way of the forehead *enables the middle ear to be circumvented more effectively than when mastoid* process is used. This is probably due to the fact that the axis of rotation of the oscillation *of the auditory ossicles (1 degree of freedom of movement) lies at an angle of* 16–24° to the median sagittal plane of the head, whereas the angle between this axis of rotation and the "*German horizontal plane*" *varies between 0* and 5° [67].

In view of these significant practical and diagnostic advantages of bone conduction through the forehead, it is undoubtedly that, as Hart and Naunton [53] have recently demonstrated, the tests made at the frontal bone are more reliable than those performed at the mastoid. Therefore the forehead can be considered as being the preferable point of application of the vibrator, even though a rise in the bone-conduction hearing threshold level of about 10 db must be accepted [8, 67, 86, 99].

In contrast to the air conduction receivers, the vibrator stimulates both cochleas with almost equal intensity [6, 8, 15, 18, 24, 25, 67, 86, 88, 115, 120, 130, 138, 143]. For this reason the elimination of one ear while measuring the bone conduction hearing threshold level of the opposite ear presents a serious problem. In order to circumvent some of the difficulties encountered in the conventional methods Rainville [113] and more recently Jerger and Tillman [60] and Lightfoot [85] have attempted to approach the measurement of bone conduction with three new procedures whose basic principles are similar. Because of their relatively great practical importance, we shall, in the following, analyse the different limitations of the classical methods (vibrator placed on the forehead and the mastoid process) and the more modern techniques recommended by the above mentioned authors.

CHAPTER III

CONVENTIONAL METHOD WITH THE VIBRATOR PLACED ON THE FOREHEAD

A TRANSMISSION PATHWAYS

Fig 7 shows schematically the different possible pathways of the test signal and the masking noise which are generated by the vibrator V and the receiver R_2 respectively, when the forehead is used for the bone conduction tests Since the vibrator is placed on the median line of the head, it is evident that the bone conducted sound stimulates both ears with the same amount of energy [$TL_{BSV(F1)} = TL_{BSV(F2)}$] provided of course the mechanism that conducts the sound waves to the inner ear is normal [48] Therefore it is essential to perform always bone conduction measurements as well as bone conduction calibrations with masking Otherwise we are testing only the better ear or determining bone conduction curves which may be influenced by binaural loudness summation effects [22, 47 147]

On the other hand the case with which the sound radiated by the vibrator may evoke a sensation of hearing by air conduction before the threshold for the bone conducted sound is reached has been much discussed in the literature [1, 22, 84, 89, 139] Accordingly reliable bone conduction audiometry is only possible if the transmission losses of the test signal in passing through the skin and the skull to the cochlea of the ear under test are at least 10 db lower than the corresponding trans

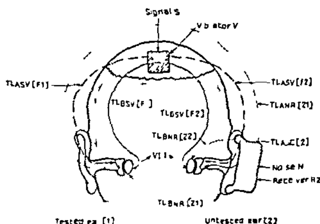


Fig 7 Schematic sketch showing the arrangement of the bone conduction vibrator V and the air-conduction receiver R_2 for measurement of the bone conduction hearing (threshold) level (Conventional method with the vibrator placed on the forehead) The various possible pathways of the test signal and the masking noise (generated by the vibrator V and the receiver R_2, respectively) are represented by different lines and arrows

mission losses through the air pathway (In this paper it will be presumed that this last requirement is fulfilled) Furthermore, seeing that at the lower frequencies the amount of the bone conduction threshold shifts due to occlusion effects may differ somewhat from one case to another (depending on the degree of the middle ear impairment), the ear under test must be uncovered in order to eliminate such sources of errors In this connection, it should however be recalled that the effective agitation reaching the organ of Corti from a bone conduction oscillator can be altered to a measurable degree by the mechanical changes in the middle ear [14 20, 21, 55, 59 62, 67, 71, 79, 84, 95, 100 106, 112, 116, 127 144, 145]

The classical procedure of masking in bone conduction measurements are based on nearly the same principles as those which are used for the determination of the air conduction curves In the following we shall, on the basis of an analysis of three hypothetical particular cases attempt to establish the limitations of masking when the vibrator is placed on the forehead

B PARTICULAR HYPOTHETICAL CASES

1 Cochleo-neural deafness

Figs 8A and B show schematically the various important factors which must be taken into consideration when one evaluates in cases of pure unilateral cochleo-neural deafness the bone conduction hearing threshold level of the diseased ear Since the untested ear is covered by an air conduction receiver, it is evident that in accordance with the graphic analysis of Fig 8B the minimum effective intensity of masking noise is given by

(8) $Imin_{N(2)} = HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)}$

In Fig 8A it is clearly shown that the masking noise may stimulate the tested ear either by bone or air conduction Taking into account that $\Delta HL_{BN(1)} = 0$ and $\Delta L_{ANC(1)} = 0$ (the tested ear being uncovered) one can easily derive from the expressions 2a and 2b the formulae for the calculation of the maximum allowable intensity of masking noise The expressions 2a and 2b become thus respectively

(9a) $Imax_{N(2)} = TL_{BNR(21)} + \Delta I_{N(1)} + HL_{BS(1)}$ and

(9b) $Imax_{N(2)} = TL_{ANR(21)} + \Delta I_{N(1)} + HL_{AS(1)}$

Finally it follows that in the cases of pure cochleo neural deafness the measurements of the bone conduction hearing threshold level for a given pure tone may be successfully carried out whenever the following conditions are fulfilled

(10a) $HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} < TL_{BNR(21)} + \Delta I_{N(1)} + HL_{BS(1)}$ and

(10b) $HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} < TL_{ANR(21)} + \Delta I_{N(1)} + HL_{AS(1)}$

Supposing for simplicity that $\Delta I_{N(1)} = \Delta I_{N(2)}$ one obtains then

(11a) $\Delta HL_{BS(2)} < TL_{BNR(21)}$ and

(11b) $\Delta HL_{BS(2)} < TL_{ANR(21)}$

Seeing that these requirements are always respected, the exclusion of the untested ear by means of a masking noise presents no serious difficulties in the cases of pure cochleo-neural deafness

placed on the forehead)
Fig 8B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

2 *Mixed deafness*

It is well known that in cases of mixed deafness the cross masking effects occur usually through the skull pathway Moreover, since the test signal which excites the organ of Corti is transmitted to the cochlea of the ear under test by bone conduction *the highest measurable bone-conduction hearing threshold level is theoretically* more or less independent of the amount of the transtympanic transmission losses of the ear under test Thus we have to distinguish only two cases

$$\text{i)}\ HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(22)}$$

Figs 9 A and B present a hypothetical case of mixed deafness where it is further supposed that the amount of the transtympanic transmission loss of the untested ear is lower than the corresponding amount of the transtemporal transmission loss According to the analysis schematically outlined in Figs 9 A and B one obtains

(12a) $Imin_N = HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)}$

Thus cross masking effects can be avoided only under the following condition

(13a) $HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(21)} + HL_{BS(1)} + \Delta I_{N(1)}$

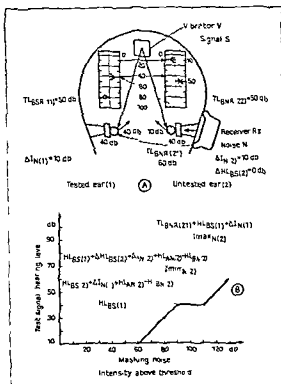


Fig 9A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the vibrator V and the receiver R_2, respectively) in the hypothetical case of a mixed deafness and where it is further supposed that $HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(22)}$ (Conventional method, with the vibrator placed on the forehead)
Fig 9B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

Assuming for simplicity that $\Delta I_{N(2)} = \Delta I_{N(1)}$ and $\Delta HL_{BS(2)} = 0$, the formula (13a) becomes

(14a) $HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(22)}$

According to this last expression it appears clearly that under the above hypothesis one can sufficiently mask the untested ear without producing cross-masking effects However the margin of safety with which the bone conduction measurements may be performed is progressively reduced as the amount of the transtympanic transmission losses of the untested ear increases

b) $HL_{AN(2)} - HL_{BN(2)} \geq TL_{BNR(22)}$

In the hypothetical case analysed in Figs 10A and B it is assumed that the amount of the transtympanic transmission loss of the untested ear is higher than the corresponding amount of the transtemporal transmission loss In similar cases, one can derive the minimum effective intensity of masking noise from the following expression

(13b) $Imin_{N(2)} = HL_{BS(1)} + \Delta I_{N(2)} + TL_{BNR(22)}$

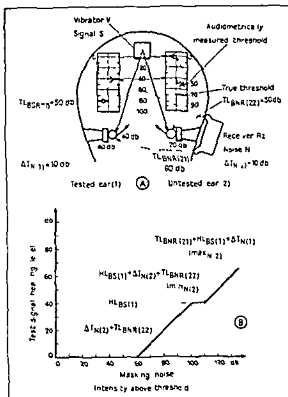


Fig 10 A
the maski
thetical ca
$- TL_{BNR(..}$
Fig 10B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

It follows that one is able to measure accurately the bone conduction hearing threshold level of the ear under test only if the following requirement is satisfied

(13b) $HL_{BS(1)} + \Delta I_{N(2)} + TL_{BNR(22)} < TL_{BNR(21)} + HL_{BS(1)} + \Delta I_{N(1)}$

Supposing that $\Delta I_{N(2)} = \Delta I_{N(1)}$ one obtains

(14b) $TL_{BNR(22)} < TL_{BNR(21)}$

Accordingly, the success with which the bone conduction curve may be recorded depends highly on the difference between the amount of the transcranial transmission loss and the amount of the transtemporal transmission loss. Since this difference is usually very small or negligeable, under the above hypothesis it is practically impossible to perform an accurate bone-conduction test

In summary the foregoing considerations lead one to draw the general conclusion that when the vibrator is applied to the forehead, the bone-conduction hearing threshold level for a given pure tone may be evaluated with a certain margin of safety only so long as the amount of the transtympanic transmission loss of the untested ear is lower than the amount of its transtemporal transmission loss

CHAPTER IV

CONVENTIONAL METHOD WITH THE VIBRATOR APPLIED TO THE MASTOID PROCESS

A TRANSMISSION PATHWAYS

Fig 11 presents schematically the various possible pathways of the test signal and the masking noise, when the mastoid process is used for the conventional bone conduction tests In general, the respective pathways of the test signal and of the masking noise are similar to those described in the preceding section However as one can see in Fig 11 the test signals which are emitted by the vibrator have a shorter route for reaching either the auditory canal or the cochlea of the ear under test than in the cases where the vibrator is placed on the forehead Although one might expect that in accordance with the inverse square law, the difference $TL_{BSV(M12\ M11)}$ between the amount of the transcranial transmission loss $TL_{BSV(M12)}$ and the amount of the transtemporal transmission loss $TL_{BSV(M11)}$ is perhaps for the lower tones as high as 10 db, there is now some evidence that at 500 c p s or less this difference is very often lower [18 25 49 67 143] In this connection it is interesting to mention the fact that in normal persons a bone-conduction oscillator placed on certain regions of the skull outside the median line may be sometimes heard in the controlateral ear and not, as commonly expected in the ipsilateral ear [24, 67, 88, 120, 130, 138] Such a phenomenon (cross perception) has usually been only observed at the lower frequency range (250 and 500 c p s) According to Weille and Gargano [143] who

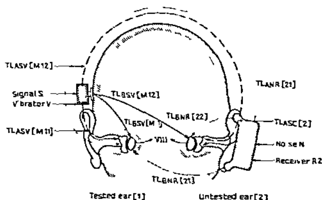


Fig 11 Schematic sketch showing the arrangement of the bone conduction vibrator V and the air conduction receiver R_2 for *measurement of the bone conduction* hearing (threshold) level (Conventional method with the vibrator placed on the mastoid) The various possible pathways of the test signal and the masking noise (generated by the vibrator V and the receiver R_2, respectively) are represented by different lines and arrows.

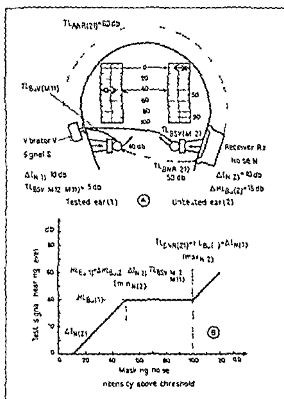


Fig 12 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the vibrator V and the receiver R_2, respectively) in the hypothetical case of a unilateral cochleo neural deafness (Conventional method, with the vibrator placed on the mastoid)
Fig 12 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

have compared the different results that one may obtain from patients suffering from unilateral complete deafness when the vibrator is alternatively applied to the two mastoid processes, the difference between the bone conduction hearing threshold levels recorded from the mastoid of the totally destroyed ear and from the normal side is sometimes at the higher frequency range (2000 to 4000 c p s) as high as 20 db However since in general one can never safely rely upon a noticeable transcranial insulation over the whole frequency range, it is obvious that masking must be routinely employed for the ear not being examined

B PARTICULAR HYPOTHETICAL CASES

1 Cochleo neural deafness

Figs 12 A and B show schematically an analysis of the different factors which must be taken into consideration in cases of pure cochleo neural deafness when one is performing bone conduction tests with the vibrator applied to the mastoid As one

Fig 13A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the vibrator V
thetical case of a bilateral mixed deafness an
$- HL_{BN(2)} < TL_{BNR(22)}$ (Conventional method,
Fig 13B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

can deduce from the graph of Fig 12B, the minimum effective intensity of masking noise is given by

(15) $\quad Imin_{N(2)} = HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} \quad TL_{BSV(M12\ M11)}$

Thus on the basis of the formulae (9a) and (9b), there is no danger for producing cross masking effects if the following conditions are satisfied

(15a) $\quad HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} \quad TL_{BSV(M12\ M11)} < TL_{BNR(21)} + \Delta I_{N(1)} + HL_{BS(1)}$ and

(15b) $\quad HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} - TL_{BSV(M12\ M11)} < TL_{ANR(21)} + \Delta I_{(N1)} + HL_{AS(1)}$

Let us assume that $\Delta I_{N(1)} \cong \Delta I_{N(2)}$ We have then

(16a) $\quad \Delta HL_{BS(2)} < TL_{BNR(21)} + TL_{BSV(M12\ M11)}$ and

(16b) $\quad \Delta HL_{BS(2)} < TL_{ANR(21)} + TL_{BSV(M12\ M11)}$

These last requirements are usually always fulfilled Therefore, in the cases of pure cochleo neural deafness one can eliminate, as expected, the untested ear without the risk of cross-masking

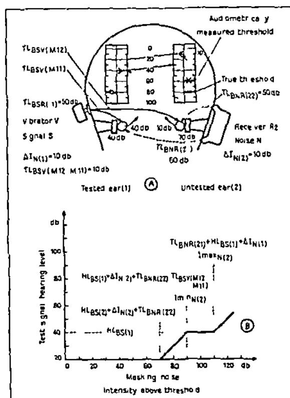


Fig 14A Schematic representation of the various important pathways of the test signal and

Fig 14B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

2 *Mixed deafness*

a) $HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(22)}$

According to the schematic analysis of Figs 13A and B, the minimum effective intensity of masking noise may be derived in the case of a mixed deafness where the amount of the transtemporal transmission loss of the untested ear is higher than the amount of its transtympanic transmission loss from the following formula

(17a) $Imin_{N(2)} = HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)} - TL_{BSV(M12\ M11)}$

It follows that one can mask sufficiently the untested ear without producing cross-masking effects, whenever

(18a) $HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)} + HL_{AN(2)} - HL_{BN(2)} - TL_{BSV(M12\ M11)} < TL_{BNR(21)} + HL_{BS(1)} + \Delta I_{N(1)}$

Supposing that $\Delta I_{N(1)} = \Delta I_{N(2)}$ and $\Delta HL_{BS(2)} = 0$, one obtains finally

(19a) $HL_{AN(2)} - HL_{BN(2)} < TL_{BNR(21)} + TL_{BSV(M12\ M11)}$

By comparing this last expression with the expression (14a), it appears clearly that, when the vibrator is placed on the forehead, the margin of safety with which the bone-conduction test can be performed at a given frequency is lower than in the above case only if the difference $[TL_{BNN(M12-M11)}]$ between the transcranial transmission losses and the transtemporal transmission losses is noticeable

$$\text{b)}\ HL_{AN(2)} - HL_{BN(2)} \gtrsim TL_{BNR(22)}$$

Such a particular case is analysed in Figs 14A and B Accordingly, *the minimum* effective intensity of masking noise can be *calculated from the following formula*

(17b) $\quad Imin_{N(2)} = HL_{BS(1)} + \Delta I_{N(2)} + TL_{BNR(22)} - TL_{BSN(M12\ M11)}$

Thus, cross masking effects may be avoided only if the following condition is satisfied

(18b) $\quad HL_{BS(1)} + \Delta I_{N(2)} + TL_{BNR(22)} - TL_{BSN(M12\ M11)} < TL_{BNR(21)} + HL_{BS(1)} + \Delta I_{N(1)}$

On the assumption that $\Delta I_{N(2)} = \Delta I_{N(1)}$, *one obtains then*

(19b) $\quad TL_{BNR(22)} < TL_{BNR(21)} + TL_{BSN(M12\ M11)}$

According to our foregoing considerations, it may happen that at the lower frequency range we have $TL_{BNR(22)} \cong TL_{BNR(21)}$ and $TL_{BSN(M12\ M11)} \cong 0$
Therefore, as in the case where the bone conduction tests are made at the frontal bone, the margin of safety with which one can circumvent cross masking effects is practically insignificant, when the amount of the transtympanic transmission loss of the untested ear is higher than/or equal to the amount of its transtemporal transmission loss

C GENERAL CONCLUSIONS AND COMMENTS

On the basis of the preceding analyses the following general conclusions should be emphasized

a) If $HL_{BS(1)} \gtrsim HL_{BS(2)}$, one is unable to determine correctly with a reasonable margin of safety the bone conduction threshold level for the test signal of the ear under examination $[HL_{BS(1)}]$, without delivering a masking noise to the untested ear, even when the vibrator is applied to the mastoid process

b) Theoretically for a given test signal (provided the minimum intensity of masking noise and/or the bone conduction hearing treshold level of the tested ear do not exceed the maximum intensity limit of the audiometer) the measurement of the bone conduction hearing threshold level of the ear with the poorer bone conduction *may be successfully carried out* (regardless of the amount of the transtympanic transmission losses of that ear), *if the amount of the* transtympanic transmission losses of the untested ear *is smaller than the amount of its* transtemporal transmission losses

c) In the cases where, for a given test signal, the *amount of the transtympanic* transmission losses of the untested ear is higher than/or equal *to the amount of its* transtemporal transmission losses, it is impossible (provided the *bone conduction of the* tested ear is poorer than that of the untested ear) to estimate the *bone-conduction* hearing threshold level of the ear under examination, without producing *cross mask*

ing effects if the amount of the transtemporal transmission loss is approximately equal to the amount of the transcranial transmission loss.

Usually one does not know which ear has the poorer bone conduction. In order to help in the selection of which ears require masking, Markle and Fowler [93] and later on, a relatively large number of authors have recommended the Weber test. However, although this test has been used for a long time in otology as a means of differentiation between cochleo-neural and middle ear deafness, Fournier and Ranville [35, 37, 39] have recently drawn our attention to the curious fact that in some cases of unilateral mixed deafness a test tone of low frequency is often lateralized toward the ear with the poorer bone conduction. According to Langenbeck [77, 78] and Legouix and Tarab [79, 134, 135] *the lateralization of the sound source to the side affected by a middle ear alteration may be attributed to an advance of phase of the sound waves in the labyrinth of the impaired ear*. Thus, seeing that in general one cannot rely upon the Weber test, it is evident that a masking noise must always be routinely applied to the untested ear when one is performing the bone conduction tests.

D. RATIONAL PROCEDURE FOR BONE CONDUCTION TESTING

For many years we have been using routinely at the University Clinic for Oto-Rhino-Laryngology of Basel a rational method [91, 92, 148] which permits determination of the bone conduction hearing threshold level without too much loss of time and degraded reliability. This masking procedure takes into account the foregoing theoretical deductions and the technical difficulties usually encountered in clinical audiometry.

1. After having recorded the air conduction curves by using the method described in the preceding section, one determines at first the bone conduction curve of the ear whose air conduction hearing threshold level is the higher. By means of the intensity potentiometer of the audiometer, one attempts to estimate the bone conduction hearing threshold level for the test signal. At the same time a narrow band of noise which is automatically matched to the frequency of the test tone, is delivered to the untested ear. Furthermore, the intensity of the masking noise is also automatically set up to the level $[HL_{BS(1)} + \Delta HL_{BS(2)} + \Delta I_{N(2)}]$ at which the test signal in the untested ear is just masked when the latter ear is normal or suffering from a pure cochleo-neural deafness. The results of this first examination may be interpreted as follows:

a) The measured bone conduction hearing threshold can be attributed to the ear under test if its level is equal to/or higher than the air conduction hearing threshold level of that tested ear.

b) In the cases where the measured bone conduction hearing threshold level is lower than the air conduction hearing threshold level of the ear under test, it is possible that the test tone is perceived in the untested ear. (In fact, the automatic masking procedure is unsatisfactory in the cases where the untested ear suffers from a mixed or a pure middle ear deafness, when the bone conduction of the ear under test is poorer than the bone conduction of the untested ear.)

2 In order to exclude this last possibility (b) in a second trial one estimates again the bone conduction after having previously raised by means of an auxiliary hand regulator the sound pressure level of the masking noise by an amount equal to the difference between the air conduction hearing threshold level of the untested ear and the bone-conduction hearing threshold level which has been determined in the course of the first investigation Thus in this second test the possible errors due to middle ear impairments of the untested ear are practically eliminated If the results of both tests are identical in the limits of the unavoidable disparities the first measurement of the bone conduction was then correct Otherwise if one has found with the second trial a higher hearing threshold level for the test signal the true bone-conduction hearing threshold level of the tested ear is given by the second measurement However because of certain individual variabilities inherent in treshold measurements it is recommended to check quickly the result of the measurements by increasing with the auxiliary hand regulator the masking noise intensity by a further amount of 5 to 15 db

3 In the cases where the result of the measurements appears inconsistent or doubtfull (cross masking effects) one performs a control test by raising in 5 db increments (in accordance with our schematic analyses) the masking noise intensity until the point is reached at which the test signal hearing threshold level remains unchanged

4 Finally on the basis of the air and bone conduction curves of one ear it is then possible by means of the hand regulator to balance out immediately the transtympanic transmission losses of the masking noise in an accurate manner when one is determining the bone conduction hearing threshold level of the other ear

As it appears from the above description this rational method enables the examiner to evaluate bone conduction curves carefully and critically without relying on the patient s ability to lateralize the test tone Needless to say that in order to facilitate the air conduction tests the masking noise intensity delivered by the audiometer used in our clinic (Jaquet audiometer) is also automatically adjusted to the level $[HL_{AS(1)}$ $TL_{BSR(12)} + \Delta HL_{BS(\,)} + \Delta I_{N(?)}]$ at which the test signal in the untested ear is just sufficiently masked when this ear is normal or suffering from a pure cochleo-neural deafness Thus in a similar manner as outlined above one must only balance out the transtympanic transmission losses of the masking noise by means of the hand regulator However in contrast to the bone conduction measurements one usually tests at first the ear with the better air conduction because of the fact that no masking is needed when the air conduction hearing threshold level of the ear under test is lower than the effective amount of the transcranial transmission losses $[TL_{BSR(1\,)}$ $\Delta HL_{B(?)}]$ and the total amount of the pericranial transmission losses $[TL_{ASR(1?)} + TL_{ASC(\,)}]$

CHAPTER V

RAINVILLE PROCEDURE

Fig 15 shows schematically the arrangement of the transducers which is employed for performing the Rainville test In this procedure the bone conduction hearing threshold level may be evaluated only after having carried out the following step-by step measurements

Step 1 —One determines the air conduction hearing threshold level for the test signal of the ear under test [$HL_{AS(1)}$]

Step 2 —The masking noise is mixed with the test tone in the earphone and gradually raised up to the level at which the air conducted test tone at threshold intensity is just masked It follows that the factor $\Delta I_{N(1)}$ can be evaluated by calculating the difference between the air conducted noise level required to mask the air conducted test tone [$HL_{AS(1)} + \Delta I_{N(1)}$] and the air conduction hearing threshold level [$HL_{AS(1)}$]

Step 3 —The same masking noise is introduced into the vibrator and gradually increased until the noise just masks the air conducted test tone at threshold intensity In other words one determines the value of the expression [$HL_{BN(1)} + \Delta I_{N(1)}$ – [illegible] the so [illegible] the ear under test [$HL_{BS(1)} - \Delta HL_{BS(1)}$] may be then estimated by determining the difference between the bone conducted noise level required to mask the test tone (generated by the air conduction receiver) and the factor $\Delta I_{N(1)}$ [$HL_{BN(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)} - \Delta I_{N(1)} = HL_{BN(1)} - \Delta HL_{BN(1)} = HL_{LS(1)} - \Delta HL_{BS(1)}$]

A PARTICULAR HYPOTHETICAL CASES

By considering the different possible pathways represented in Fig 15 it is evident that the margin of safety with which one can determine by means of the Rainville test the bone conduction hearing threshold level of the ear under test depends somewhat on the amount of the transcranial and pericranial transmission losses of the test signal In the following hypothetical examples we shall presume that for simplicity the amount of the transcranial transmission losses of the test signal [$TL_{BSR(12)}$] is lower than the amount of the pericranial transmission losses [$TL_{ASR(12)}$]

1 Cochleo neural deafness

a) $HL_{AS(1)} \quad TL_{BSR(12)} < HL_{BS(2)}$

Figs 16A and B allow us to analyse an hypothetical case of a unilateral pure cochleo neural deafness where it is further supposed that the air conduction hearing

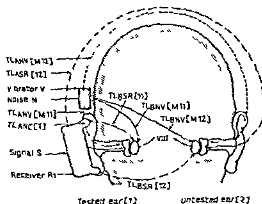


Fig 15. Schematic sketch showing the arrangement of the bone conduction vibrator V and the air conduction receiver R_1 for measurement of the bone conduction hearing (threshold) level (Rainville's technique). The various possible pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) are represented by different lines and arrows

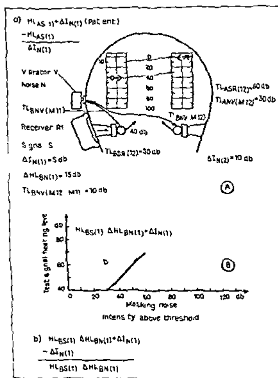


Fig 16 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a unilateral cochleo-neural deafness, and where it is further supposed that $HL_{AS(1)} < TL_{BSR(12)}$ (Rainville's technique)

Fig 16 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

threshold level for the test tone of the poorer ear is lower than the amount of the transcranial transmission losses $TL_{BSR(12)}$ According to the study of the variations of the test *signal hearing level as a function of the intensity of the masking noise* generated by the vibrator (see the graph of Fig 16B) as soon as the intensity of the masking noise reaches the level at which the test signal is just masked any further increments of the noise intensity produce an equal shift of the test signal hearing level When one estimates the absolute bone conduction hearing threshold level by following the recommendations of Rainville the limit of errors for a given test tone depends highly on the accuracy with which the examiner can determine the change over point $[HL_{BS(1)} - \Delta HL_{BN(1)} + \Delta I_{N(1)}]$ where the air conduction hearing threshold level for the test tone of the ear under test begins to be elevated From the schematic analysis and the symbolic calculations (a and b) of Figs 16A and B one can deduce that *in general the Rainville procedure may* be carried out without any difficulties in the cases of pure cochleo neural deafness, if for a given test signal the bone conduction hearing threshold level of the untested ear is higher than the difference between the air-conduction hearing threshold level of the ear under test and the amount of the transcranial transmission loss $[TL_{BSR(12)}]$

b) $HL_{AS(1)} - TL_{BSR(12)} > HL_{BS(2)}$

Fig 17A shows an hypothetical case of a unilateral pure cochleo neural deafness where the results obtained by the Rainville test may be falsified by shadow hearing effects of the test signal According to Figs 17 A and B the test signal hearing level which is measured in the first step will be equal to the amount of the transcranial transmission loss of the test signal $[TL_{BSR(12)} = 50$ db$]$ if the examiner fails to mask sufficiently the untested ear Consequently at a test signal level of 50 db (shadow *hearing) one must raise the intensity of the masking noise generated by the air* conduction receiver at least as high as $TL_{BNR(12)} + \Delta I_{N(2)}$ in order to be able to just mask the test signal (step 2) Under the assumption that $TL_{BNR(12)} = TL_{BSR(12)}$ the difference between the noise level $TL_{BNR(12)} + \Delta I_{N(2)}$ and the above mentioned test signal level $[TL_{BSR(12)} = 50$ db$]$ gives then $\Delta I_{N(2)}$ (see the symbolic calculations 1a of Fig 17A) Finally (step 3) by masking the test signal (at threshold intensity) with the aid of the noise generated by the vibrator *(change over point 1b)* one determines the amount of the sum $TL_{BNV(M12\ M11)} + \Delta I_{N(2)}$ The false measured bone conduction hearing threshold level is then as low as the amount of the factor $TL_{BNV(M12\ M11)}$ (see the symbolic calculations 1b of Fig 17B) Thus in the cases of pure cochleo neural deafness, where the *bone conduction hearing threshold* level of the untested ear is lower than the difference between the air conduction hearing threshold level of the ear under test and the amount of the transcranial transmission loss the bone conduction hearing threshold level determined by the Rainville test may be completely erroneous if the air conduction hearing threshold level for the test signal of the ear under test has not been measured by applying an adequate masking noise to the untested ear

Referring to the graph of Fig 17B it is interesting to note the presence of a plateau The level of this plateau is equal to the true air-conduction hearing threshold level

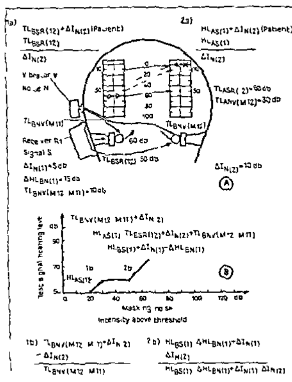


Fig 17 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a unilateral cochleo neural deafness, and where it is further supposed that $HL_{AS(1)} > TL_{BSR(12)}$ (Rainville s technique)
Fig 17 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

of the ear under test Accordingly, the bone conducted masking noise begins to mask the test signal in the ear under test, when its intensity is raised up to a level equal to $HL_{BS(1)} + \Delta I_{N(1)} \quad \Delta HL_{BN(1)}$ From this fact it is evident that [provided $\Delta I_{N(1)}$ and $\Delta I_{N(2)}$ are equal] *in the cases of pure cochleo-neural deafness the absolute bone-conduction hearing threshold level for the test signal of the poorer ear can be estimated correctly by means of the procedure proposed by Rainville, if the examiner at first carefully determines the air conduction hearing threshold level of the ear to be examined and maintains then the test signal intensity on that latter level during the performance of all the other different testing steps* (see the symbolic calculation 2a and 2b of Figs 17 A and B)

2 *Mixed deafness*

a) $HL_{AS(1)} - TL_{BSR(12)} \leq HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} > TL_{BSR(22)}$

Fig 18 A shows an hypothetical case of mixed deafness where it is impossible with the aid of the conventional methods to eliminate the untested ear without producing

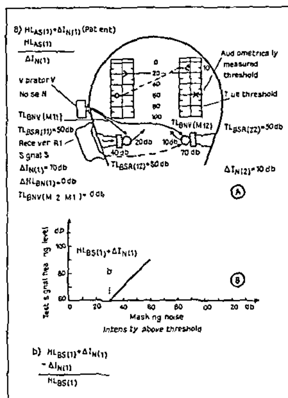


Fig 18A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a bilateral mixed deafness, and where it is further supposed that $HL_{AS(1)} - TL_{BSR(12)} = HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} > TL_{BSR(12)}$ (Rainville s technique)
Fig 18B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

cross masking effects, whereas one can successfully use the Rainville procedure for bone conduction testing However, it should be noted that, in the cases where $HL_{AS(1)} - TL_{BSR(12)} = HL_{BS(2)}$ or $HL_{BS(1)} + TL_{BSR(11)} - TL_{BSR(12)} = HL_{BS(2)}$ (depending on whether the transtympanic transmission losses of the ear under test are lower or higher than its transtemporal transmission losses), the results of the Rainville test are correct only if the following requirement is fulfilled $HL_{BS(2)} + TL_{BNV(M12\ M11)} + \Delta I_{N(2)} \leq HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)}$ In fact, when this last condition is not satisfied the bone conducted noise level just necessary for masking the test tone is as high as the amount of the sum $HL_{BS(2)} + \Delta I_{N(2)} + TL_{BNV(M12\ M11)}$ (step 3) The false measured bone conduction hearing threshold level for the test tone is then equal to the amount of the factor $HL_{BS(2)} + TL_{BNV(M12\ M11)}$

According to the specifications of Fig 18A it has been supposed that because of the severe middle ear lesions there is no bone conduction threshold shift for the masking noise from placing the air conduction receiver over the ear under test Thus, by performing the Rainville test in such a case (as one can deduce from the schematic analysis of Figs 18A and B) the examiner may underestimate the cochlear reserve

Fig 19 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a bilateral mixed deafness, and where it is further supposed that $HL_{AS(1)} - TL_{BSR(12)} > HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} < TL_{BSR(22)}$ (Rainville's technique).
Fig 19 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise intensity

at the lower frequencies [the measured bone-conduction hearing level being equal to $HL_{BS(1)}$] On the other hand, seeing that in certain cases of middle ear deafness (secretory otitis) the bone conduction is improved thanks to the occlusion effect due to the presence of a fluid in the tympanic cavity behind an intact drum [100], one is unable to detect such a phenomenon by covering up the ear under test. Therefore, if one adopts the procedure proposed by Rainville, the otologist who reads the audiogram must not only interpret the occlusion effects caused by the earphone from one

b) $HL_{AS(1)} - TL_{BSR(12)} > HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} < TL_{BSR(22)}$

Figs 19 A and B illustrate an hypothetical case of mixed deafness where the evaluation of the bone conduction hearing threshold level for the test signal of the ear with the poorer bone conduction may be incorrect because of the presence of shadow hearing effects, if the Rainville test is not carried out with some precautions.

Referring to the analysis and the symbolic calculations (1a and 1b) of Figs. 19A and B, the false bone conduction hearing threshold level, that one obtains in the case where the untested ear has not been sufficiently masked by performing the air conduction measurement (step 1) is as low as the amount of the sum $TL_{BNV(M12-M11)} + HL_{BS(2)}$.

In contrast to the hypothetical case analysed in Figs. 17A and B, it has been assumed that $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)} = HL_{AS(1)} - TL_{BSR(12)} + \Delta I_{N(2)} + TL_{BNV(M12-M11)}$. Thus one is unable to observe a plateau (i.e. the level at which the test signal is just perceived in the ear under test) by studying the variations of the hearing threshold level for the test signal as a function of the intensity of the masking noise emitted by the vibrator (see Fig. 19B). However, in such a particular case the errors caused by the shadow hearing effects of the test tone can be avoided if one has at first (step 1) carefully estimated the level at which the test signal (generated by the air conduction receiver) just evokes a sensation of hearing in the ear being examined (see the symbolic calculations 2a and 2b of Figs. 19A and B). Since it has been supposed that the bone conduction threshold shift due to the occlusion of the meatus by the earphone is negligeable (high degree of middle ear deafness) the measured bone conduction hearing threshold level is also, as in the foregoing example, equal to $HL_{BS(1)}$.

B. DISCUSSION AND CONCLUSION

In the procedure proposed by Rainville the margin of safety of the bone conduction measurements depends highly on the success with which the air conduction hearing threshold level for the test tone of the ear under examination can be evaluated (step 1). Thus, in the cases where for a given test tone the transtympanic transmission losses of both ears are higher than/or equal to the transtemporal transmission losses it is impossible (provided the bone conduction of the tested ear is poorer than that of the untested ear) to determine the true bone conduction hearing threshold level of the ear under test if the transtemporal transmission losses are approximately equal to the transcranial transmission losses $TL_{BSR(12)}$. Furthermore, by considering the last three hypothetical cases we are lead to the conclusion that, in the cases where $HL_{AS(1)} - TL_{BSR(12)} \geq HL_{BS(2)}$ or $HL_{BS(1)} + TL_{BSR(11)} - TL_{BSR(12)} \geq HL_{BS(2)}$ (depending on whether the transtympanic transmission losses of the ear under test are lower or higher than the ipsilateral transtemporal transmission losses) one is able to estimate correctly the bone conduction hearing threshold level of the ear under test only when the following requirements are fulfilled (provided the test signal in the untested ear is effectively excluded during the performance of the measurements of the air conduction hearing threshold level of the ear under test):

$HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)} \geq HL_{AS(1)} - TL_{BSR(12)} + \Delta I_{N(2)} + TL_{BNV(M12-M11)}$ or $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)} \geq HL_{BS(1)} + TL_{BSR(11)} - TL_{BSR(12)} + \Delta I_{N(2)} + TL_{BNV(M12-M11)}$

[Otherwise the false measured bone conduction hearing threshold level is as high as the amount of the factor $HL_{AS(1)} - TL_{BSR(12)} + TL_{BNV(M12-M11)}$ or of the factor $HL_{BS(1)} + TL_{BSR(11)} - TL_{BSR(12)} + TL_{BNV(M12-M11)}$]

In the cases of pure cochleo-neural deafness one determines the absolute bone conduction hearing threshold level [$HL_{BS(1)}$ $\Delta HL_{BS(1)}$] As discussed above in cases of middle ear deafness the improvement of the bone-conduction due to occlusion effects (by the earphone or fluid in the tympanic cavity) varies from one case to another Therefore in contrast to the conventional methods the accuracy of the Rainville test is less and certain subtle clues which might otherwise contribute to finer diagnostic differentiation are obscured

CHAPTER VI

RAINVILLE TEST AS MODIFIED BY LIGHTFOOT

Fig 20 shows the arrangement of the transducers to be used if one follows the recommendations of Lightfoot [85] In contrast to the Rainville test, the vibrator which generates the masking noise is placed on the forehead Moreover, before starting the tests with the patients one must at first carry out the following two steps measurements by a few young adult subjects with normal hearing, in order to get the normative data

Step 1 —One determines the air conduction hearing threshold level for the test signal of the ear under test [$HL_{AS(1)}$]

Step 2 —The masking noise is introduced into the vibrator and gradually raised up to the level at which the air conducted test tone at threshold intensity is just masked In other words, [provided that $HL_{BN(1)} = HL_{BS(1)} = HL_{AS(1)}$], one determines the value of the expression $HL_{AS(1)} + \Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ By subtracting $HL_{AS(1)}$ from this last expression, one obtains the factor $\Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ The average amounts of this factor for the different test tones then serve as the normative data

The technique used for testing the patients is the same as that described above For each test frequency one simply estimates the level at which the bone conducted masking noise just masks the pure tone (generated by the air conduction receiver) at threshold intensity [i e one determines the amount of the expression $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)}$] Thus under the assumption that $\Delta HL_{BN(1)} = \Delta HL^*_{BN(1)}$ and $\Delta I_{N(1)} = \Delta I^*_{N(1)}$ one obtains for a given test tone the so called relative bone conduction hearing threshold level $HL_{BS(1)}$ (patient) by subtracting the corresponding average value of the factor $\Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ (normal subject) from the amount of the expression $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)}$

A PARTICULAR HYPOTHETICAL CASES

1 Cochleo neural deafness

Under the hypothesis that the transmission losses of the test signal in passing through the air pathway to the untested ear are higher than the transcranial transmission losses $TL_{BSR(12)}$ we must distinguish as in the preceding section the following two cases

$$a)\ HL_{AS(1)} - TL_{BSR(12)} < HL_{BS(2)}$$

Figs 21 A and B allow us to analyse the hypothetical case of a unilateral pure cochleo neural deafness where it is further supposed that the air conduction hearing threshold level for the test tone of the poorer ear is lower than the amount of the trans

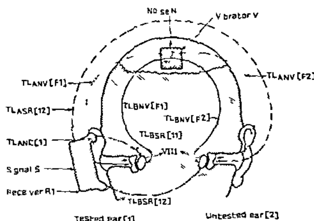


Fig 20 Schematic sketch showing the arrangement of the bone conduction vibrator V and the air conduction receiver R_1 for measurement of the bone conduction hearing (threshold) level (Modification of Rainville s technique)

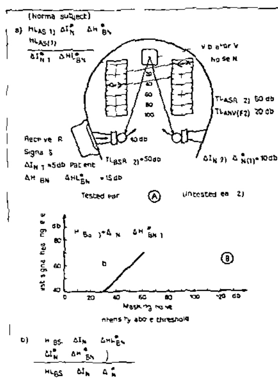


Fig 21 A Schematic representation of the various important pathways of the test signal and the mask
thetical c
$HL_{AS(1)}$
Fig 21 B
test signal as a function of the masking noise intensity

CHAPTER VI

RAINVILLE TEST AS MODIFIED BY LIGHTFOOT

Fig 20 shows the arrangement of the transducers to be used if one follows the recommendations of Lightfoot [85] *In contrast to the Rainville test, the vibrator* which generates the masking noise is placed on the forehead Moreover before starting the tests with the patients, one must at first carry out the following two steps measurements by a few young adult subjects with normal hearing, in order to get the normative data

Step 1 —One determines the air conduction hearing threshold level for the test signal of the ear under test [$HL_{AS(1)}$]

Step 2 —The masking noise is introduced into the vibrator and gradually raised up to the level at which the air conducted test tone at threshold intensity is just masked In other words, [provided that $HL_{BN(1)} = HL_{BS(1)} = HL_{AS(1)}$], one determines the value of the expression $HL_{AS(1)} + \Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ By subtracting $HL_{AS(1)}$ from this last expression one obtains the factor $\Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ The average amounts of this factor for the different test tones then serve as the normative data

The technique used for testing the patients is the same as that described above For each test frequency one simply estimates the level at which the bone conducted masking noise just masks the pure tone (generated by the air conduction receiver) at threshold intensity (i e one determines the amount of the expression $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)}$) Thus under the assumption that $\Delta HL_{BN(1)} = \Delta HL^*_{BN(1)}$ and $\Delta I_{N(1)} = \Delta I^*_{N(1)}$ one obtains for a given test tone the so called relative bone conduction hearing threshold level $HL_{BS(1)}$ (patient) by subtracting the corresponding average value of the factor $\Delta I^*_{N(1)} - \Delta HL^*_{BN(1)}$ (normal subject) from the amount of the expression $HL_{BS(1)} + \Delta I_{N(1)} - \Delta HL_{BN(1)}$

A PARTICULAR HYPOTHETICAL CASES

1 Cochleo neural deafness

Under the hypothesis that the transmission losses of the test signal in passing through the air pathway to the untested ear are higher than the transcranial transmission losses $TL_{BSR(12)}$ we must distinguish as in the preceding section the following two cases

a) $HL_{AS(1)} - TL_{BSR(12)} < HL_{BS(2)}$

Figs 21 A and B allow us to analyse the hypothetical case of a unilateral pure *cochleo neural deafness* where it is further supposed that the air conduction hearing threshold level for the test tone of the poorer ear is lower than the amount of the trans

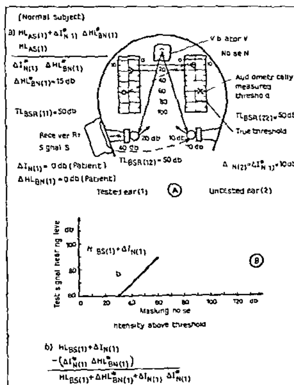


Fig 23 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a bilateral mixed deafness, and where it is further supposed that $HL_{AS(1)} - TL_{BSR(12)} = HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} > TL_{BSR(22)}$ (Modification of Rainville's technique)

Fig 23 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise

applying a masking noise to the better ear the false measured bone conduction hearing threshold level of the poorer ear is then equal to $\Delta I_{N(2)} \quad \Delta I^*_{N(1)} + \Delta HL^*_{BN(1)}$ (see the symbolic calculations 1 b of Fig 22 B)

In view of the schematic analysis of Fig 22 B (provided $\Delta I_{N(1)}$ and $\Delta I^*_{N(1)}$ are equal), it is evident that in the cases of pure cochleo neural deafness, one can evaluate correctly the relative bone conduction hearing threshold level of the poorer ear (see the symbolic calculations 2 b of Fig 22 B) whenever the air conduction tests of that ear have been performed with sufficient care

2 *Mixed deafness*

a) $HL_{AS(1)} - TL_{BSR(12)} \leq HL_{BS(2)}$ and $HL_{AS(2)} \quad HL_{BS(2)} > TL_{BSR(22)}$

Figs 23 A and B allow us to analyse an hypothetical case of mixed deafness where it is impossible with the conventional methods to eliminate the untested ear without producing cross masking effects In the example under consideration, it is assumed

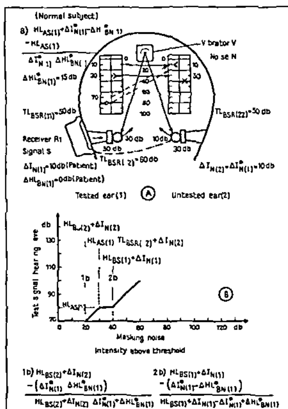


Fig 24 A Schematic representation of the various important pathways of the test signal and the masking noise (generated by the receiver R_1 and the vibrator V, respectively) in the hypothetical case of a bilateral mixed deafness and where it is further supposed that $HL_{AS(1)}$ $TL_{BSR(12)} > HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} < TL_{BSR(22)}$ (Modification of Rainville s technique)

Fig 24 B Schematic representation of the variations of the hearing (threshold) level for the test signal as a function of the masking noise

that because of the severe middle ear lesions, there is no bone conduction threshold shift for the masking noise from placing the air conduction receiver over the tested ear, whereas the normal bone conduction threshold shift $\Delta HL^*_{BN(1)}$ is as high as 15 db Thus, in the cases of middle ear deafness the examiner may underestimate

foot the occlusion effects due to the presence of a fluid in the tympanic cavity

b) $HL_{AS(1)} - TL_{BSR(12)} > HL_{BS(2)}$ and $HL_{AS(2)} - HL_{BS(2)} < TL_{BSR(22)}$

Figs 24 A and B show an hypothetical case of mixed deafness where the results obtained with the Rainville test as modified by Lightfoot may be erroneous, whereas the conventional procedure does not present particular difficulties Under the assumption that the air conduction test of the worse bone conducting ear has been

carried out without applying a masking noise to the untested ear the false measured bone conduction hearing threshold level [provided $\Delta I_{N(2)} = \Delta I^{*}_{N(1)}$] is then equal to $HL_{BS(2)} + \Delta HL^{*}_{BN(1)}$ (see the symbolic calculations 1b of Fig 24B) Furthermore according to the specifications of Fig 24 A the bone-conduction threshold shift for the masking noise from placing the air-conduction receiver over the tested ear (patient) is negligeable Thus even though the air-conduction hearing threshold level of the ear under test has been correctly determined from the analysis of Fig 24B it appears that in such a case one underestimates the cochlear reserve of the ear under test by using the Rainville test as modified by Lightfoot

B CONCLUSIONS

Although in contrast to the Rainville test the new technique proposed by Lightfoot is a less time consuming method the above analyses show that the application of both procedures *are associated with nearly the same difficulties* Thus for a given test tone the accuracy of the bone conduction measurements performed with the aid of the Rainville test as modified by Lightfoot depends on the success with which the air conduction hearing threshold level has been evaluated Moreover despite the fact that theoretically one determines the relative bone-conduction hearing threshold level $HL_{BS(1)}$ by using the Rainville test as modified by Lightfoot in cases of middle ear deafness the results of the measurements must be interpreted as in the Rainville test from one case to another with great care because of the variations of the occlusion effect (by the air conduction receiver or fluid in the tympanic cavity) However the bone conducted masking noise stimulates both ears with the same amount of energy [$TL_{BNV(F1)} = TL_{BNV(F2)}$] when one employs the procedure outlined by Lightfoot

losses) it is possible to estimate correctly [under the condition that $\Delta I^{*}_{N(1)}$ $\Delta I_{N(1)}$ $= \Delta I_{N(2)}$ and provided one can carry out the air conduction measurements without

It has been shown that, for a particular pure tone and a given transcranial transmission loss, the margin of safety with which the air conduction tests can be performed without producing cross masking effects diminishes as the sum of the transtympanic transmission losses of both ears increases On the other hand, regardless of the amount of the transtympanic transmission loss of the ear under examination, the bone conduction hearing threshold level of this ear may be correctly estimated by the conventional method, so long as the amount of the transtympanic transmission loss of the untested ear is lower than the amount of its transtemporal transmission loss Undoubtedly, in the relatively infrequent cases where the transtympanic transmission losses of the untested side are higher or approximately equal to the transtemporal transmission losses the more modern methods as proposed by Rainville, Lightfoot, Jerger and Tillman may be sometimes very helpful for the evaluation of the cochlear reserve of the ear under examination However, seeing that the results of the measurements obtained with these new procedures are dependent on the accuracy with which the air conduction hearing threshold levels have been evaluated and may, furthermore, be obscured by the unpredicable bone conduction threshold shifts due to the occlusion effect (by the air conduction receiver or fluid in the tympanic cavity), the conventional method still seems to be the preferable procedure for the clinical purposes In fact, one must hope that in the near future the manufactors will make a greater effort for providing audiometers with air conduction receivers that will improve the transtemporal and transcranial insulation Thus not only will the need to mask the untested ear in air conduction tests be almost eliminated, but the danger of cross masking effects in conventional bone conduction measurements will also be reduced

SUMMARY

The principal objective of the present study was to discuss the limitations of masking by conventional audiometry and the new procedures recommended by Rainville, Lightfoot, Jerger and Tillman The author recalls at first briefly the principal advantages of narrow band over broad band noise, as well as the general rules governing the practical application of masking in clinical audiometry The limits within which the intensity of the masking noise must be maintained in order to exclude efficiently the test tone in the untested ear without fear of cross masking effects are studied on the basis of a detailed analysis of some hypothetical examples It is shown that, for a particular test tone and a given transcranial transmission loss, one is only able to determine accurately the air conduction hearing threshold level of the ear under examination so long as the transtympanic transmission losses of this ear are lower than its transtemporal transmission losses, furthermore, the margin of safety with which the unwanted ear can be blocked during the air-conduction tests diminishes as the sum of the transtympanic transmission losses of both ears increases

The reasons why placing the vibrator on the forehead is much more satisfactory for the determination of the bone-conduction hearing threshold level than applying the vibrator to the mastoid process are reviewed From our analyses and symbolic calculations it appears that for a given test tone, one may in general be able to estimate correctly the cochlear reserve of the poorer ear by using the conventional method (with the vibrator placed on the forehead or the mastoid process) whenever the amount of the transtympanic transmission loss of the better ear is lower than the amount of its transtemporal transmission loss In order to minimize certain inconveniences usually associated with the conventional method a rational procedure is recommended which allows us to record the audiograms without too much loss of time and degraded reliability

Finally the limitations of the methods recently proposed by Rainville Lightfoot Jerger and Tillman are also clarified by means of some hypothetical examples It is shown that the accuracy of the bone conduction measurements carried out with the aid of these methods depends highly on the success with which the air-conduction hearing threshold level for the test tone of the ear under examination can be evaluated, furthermore because of the unpredicable occlusion effects (by the air conduction receiver or fluid in the tympanic cavity) the results of the measurements must be interpreted with great care in cases of middle ear deafness

ZUSAMMENFASSUNG

Diese Arbeit wurde in der Absicht geschrieben die Grenzen der klassischen Audiometrie und der von Rainville Lightfoot Jerger und Tillman vorgeschlagenen neuen Methoden festzustellen Der Autor weist kurz auf die hauptsachlichen Vorteile des Schmalbandgerausches gegenuber dem Breitbandgerausch hin ebenso auf die Grundprinzipien des Vertaubungsverfahrens bei klinischen Horprufungen Der Bereich in *welchem die Vertaubungslautstarke gehalten werden muß damit die ausgeloste* Horempfindung fur den Pruſton im nichtgeprüften Ohr ohne Gefahr eines «gekreuzten Maskierungseffektes» verschwindet wird auf Grund einer eingehenden Analyse einiger hypothetischer Beispiele studiert Es wird gezeigt daß es nur dann moglich ist fur einen gegebenen Pruſton und Überleitungsverlust durch den Schadel den Pegel der Luftleitungsschwelle des gepruften Ohres genau zu bestimmen wenn der Mittelohr uberleitungsverlust dieses Ohres niedriger ist als sein «Temporaluberleitungsverlust» zudem verkleinert sich die Sicherheitszone in welcher das nicht geprufte Ohr wahrend der Luftleitungsmessungen blockiert werden kann wenn die Summe der Mittelohr uberleitungsverluste beider Ohren sich vergroßert

Die Grunde weshalb die Stirne als Ansatzstelle fur den Vibrator zur Bestimmung der Knochenleitungsschwelle bedeutend vorteilhafter ist als der Warzenfortsatz werden ebenfalls erwahnt Aus unserer Analyse die Knochenleitungsmessungen betreffend geht hervor daß es dem Audiometristen im allgemeinen moglich ist die cochleare Reserve des schlechteren Ohres fur einen bestimmten Pruſton mit Hilfe des klassischen Verfahrens (Vibrator auf der Stirne oder dem Warzenfortsatz) richtig zu schätzen wenn der Mittelohruberleitungsverlust des besseren Ohres kleiner ist als der «Temporaluberleitungsverlust» Um gewisse Unannehmlichkeiten der klassischen Methode auf ein Minimum zu reduzieren wird ein rationelles Verfahren empfohlen welches erlaubt zuverlassige Audiogramme ohne zuviel Zeitverlust aufzunehmen.

Schließlich werden die Grenzen der von Rainville Lightfoot Jerger und Tillman vorgeschlagenen Methoden auch an Hand einiger hypothetischer Beispiele erlautert Es wird gezeigt daß die Genauigkeit der mit Hilfe dieser Verfahren durchgefuhrten Knochenleitungsmessungen stark von dem Erfolg mit welchem der Pegel der Luftleitungshorschwelle des untersuchten Ohres bei der Meßfrequenz bestimmt werden kann abhangig ist Wegen der durch Gehorgangsverschluß (Luftleitungshorer) oder durch die in der Paukenhohle enthaltene Flussigkeit verursachten Veranderungen der Knochenleitungsschwelle mussen zudem die Ergebnisse der Messungen in Fallen von Mittelohrschwerhorigkeiten sehr sorgfaltig interpretiert werden

RÉSUMÉ

Ce travail a été écrit dans le but de préciser les limites de l'audiométrie classique et des nouveaux procédés proposés par Rainville, Lightfoot, Jerger et Tillman L'auteur rappelle tout d'abord brièvement les principaux avantages du bruit à bande étroite par rapport au bruit à large bande, ainsi que les principes fondamentaux de l'assourdissement en audiométrie clinique En s'appuyant sur une analyse détaillée de quelques exemples hypothétiques, on établit des expressions qui permettent d'évaluer la zone dans laquelle l'intensité sonore du bruit d'assourdissement doit être maintenue afin d'éliminer, dans l'oreille non interrogée, l'audition du son test sans aucun risque de retentissement de ce son masquant sur l'oreille interrogée On montre que, pour un son test particulier et une perte de transfert transcrânienne donnée, il n'est possible de déterminer avec précision le niveau du seuil d'acuité auditive par conduction aérienne de l'oreille interrogee que dans les cas où la perte de transfert «transtympanique» de cette oreille est plus petite que sa perte de transfert «trans temporale», de plus, la marge de sécurité avec laquelle l'on peut assourdir, d'une manière adequate et rigoureuse, l'oreille non interrogée, lors du relevé des courbes aériennes, est d'autant plus petite que la somme totale des pertes de transfert «transtympanique» des deux oreilles augmente

On indique egalement les raisons pour lesquelles l utilisation du front, comme point d'application du vibrateur à conduction osseuse, est notablement plus favorable que l'emploi de la mastoïde Il decoule de notre analyse concernant la détermination de la courbe de conduction osseuse que, pour un son test donné l'audiométriste a, en general, la possibilite d'estimer correctement la réserve cochléaire de l'oreille la plus déficiente a l'aide de la methode classique (vibrateur appuyé contre le front ou la mastoide) quand la perte de transfert «transtympanique» de la meilleure oreille est plus petite que sa perte de transfert «transtemporale» Afin de réduire au minimum certains inconvenients habituellement associes avec la methode classique, on recommande un procédé rationnel qui permet de relever des audiogrammes rigoureux sans tâtonnement et avec une économie de temps significative

Finalement, on précise encore a l'aide de quelques exemples hypothetiques les limites des methodes récemment proposées par Rainville, Lightfoot, Jerger et Tillman On montre que la precision des mesures de conduction osseuse effectuées avec ces methodes dépend du succès avec lequel l'on peut determiner le niveau du seuil d acuité auditive par conduction aerienne de l oreille interrogée, de plus, a cause des variations du seuil d acuité auditive par conduction osseuse dues à l'obturation (écouteur a conduction aerienne) du canal auditif externe ou à la presence d'un fluide dans la caisse du tympan, les résultats des mesures doivent être interpretés avec un grand soin dans les cas ou la surdité est exclusivement ou partiellement provoquée par une altération de l'oreille moyenne

EXPLANATION OF THE SYMBOLS

HL	Hearing (threshold) level (expressed in decibels relative to the zero reference level of the audiometer) Niveau du seuil d'audibilité Pegel der Hörschwelle
TL	Transmission loss (expressed in decibels) Perte de transmission ou perte de transfert Überleitungsverlust
A	Air conduction Conduction aérienne Luftleitung
B	Bone conduction Conduction osseuse Knochenleitung
S	Test signal (pure tone) Son test Prüfton
N	Masking noise (narrow band of noise) Bruit d'assourdissement ou son masquant (bruit à bande étroite) Vertäubungsgeräusch (Schmalbandgeräusch)
R	Air conduction receiver Écouteur à conduction aérienne Luftleitungshörer R_1 = Air conduction receiver placed on the tested ear (1) R_2 = Air conduction receiver placed on the untested ear (2)
C	Cushion of the receiver Coussinet de l'écouteur à conduction aérienne Polster des Luftleitungshörers
V	Bone conduction vibrator Vibrateur à conduction osseuse Knochenleitungshörer
F	Forehead Front Stirne
M	Mastoid Mastoïde Mastoid (Warzenfortsatz)
$HL_{AS(1)}$	Air conduction hearing (threshold) level for the test signal of the ear under test
$HL_{AS(2)}$	Air conduction hearing (threshold) level for the test signal of the untested ear
$HL_{BS(1)}$	Bone conduction hearing (threshold) level for the test signal of the ear under test (auditory canal open)
$HL_{BS(2)}$	Bone conduction hearing (threshold) level for the test signal of the untested ear (auditory canal open)
$HL_{AN(1)}$	Air conduction hearing (threshold) level for the masking noise of the ear under test

$HL_{AN(2)}$ Air conduction hearing (threshold) level for the masking noise of the untested ear

$HL_{BN(1)}$ Bone conduction hearing (threshold) level for the masking noise of the ear under test (auditory canal open)

$HL_{BN(2)}$ Bone conduction hearing (threshold) level for the masking noise of the untested ear (auditory canal open)

$\Delta HL_{BS(1)}$ Bone conduction threshold shift (expressed in decibels) for the test signal from placing the air conduction receiver R_1 over the tested ear (patient)

$\Delta HL_{BS(2)}$

$\Delta HL_{BN(1)}$ air

$\Delta HL^*_{BN(1)}$ Bone conduction threshold shift for the masking noise from placing the air conduction receiver R_1 over a normal ear
$\Delta HL^*_{BN(1)} = HL_{BN(1)}$ open $HL_{BN(1)}$ closed
(normal ear) (normal ear)

$\Delta HL_{BN(2)}$

I_N Intensity of noise (decibels reading of the attenuator)

$\Pi_{N(2)}$ Just necessary increment above its threshold level of the intensity of noise (expressed in decibels) for masking the test signal (at threshold intensity) in the untested ear (patient)
Minimum d intensite du bruit d assourdissement (exprime en decibels au dessus du seuil d audibilite de ce son masquant) que l on doit fournir a l oreille non interrogee (2) pour faire disparaitre en celle ci la perception aerienne ou osseuse du son test prise a son seuil

$\Pi_{N(1)}$ Just necessary increment above its threshold level of the intensity of noise (expressed in decibels) for masking the test signal (at threshold intensity) in the tested ear (patient)

$\Pi^*_{N(1)}$ Just necessary increment above its threshold level of the intensity of noise (expressed in decibels) for masking the test signal (at threshold intensity) in a normal ear

$I_{minN(*)}$ Minimum effective intensity of masking noise (expressed in decibels above the normal threshold of hearing for that noise)
«Minimum d intensité efficace» du bruit d assourdissement La plus petite intensite du son masquant (exprimee en decibels au dessus du seuil normal d audibilite de ce son masquant) necessaire a l assourdissement efficace du son test dans l oreille non interrogee
Minimale Vertäubungslautstärke (notwendige Vertaubungsstarke)

$Imax_{N(2)}$ Maximum allowable intensity of masking noise (The intensity, measured in decibels above the normal threshold level of the noise, at which masking of the signal at the tested ear begins)
Maximum d intensité non retentissante du bruit d'assourdissement (exprimé en decibels au dessus du seuil normal d audibilité de ce bruit) a partir duquel le son masquant étend son effet de masque à l oreille examinée
Maximale Vertaubungslautstarke (zulässige Maskierungsstarke)

$D_{N(2)}$

decibels au dessus du seuil normal d audibilité de ce son masquant)
Schmerzschwelle fur das Vertaubungsgeräusch (in dB uber der Normalhorschwelle fur dieses Vertaubungsgerausch)

$TL_{ASC(2)}$ Transmission loss of the test signal (radiated by the receiver R_1 or the vibrator V) in passing through the cushion of the receiver R_2

$TL_{ANC(1)}$ Transmission loss of the masking noise (radiated by the receiver R_2 or the vibrator V) in passing through the cushion of the receiver R_1

$TL_{ASR(12)}$

untested ear being open)

$TL_{ANR(21)}$

the ear under test being open)

$TL_{BSR(11)}$ Transmission loss (transtemporal attenuation) of the test signal (generated by the receiver R_1) in passing through the temporal bone to the cochlea of the tested ear

$TL_{BSR(12)}$ Transmission loss (transcranial attenuation) of the test signal (generated by the receiver R_1) in passing through the skull to the cochlea of the untested ear (the auditory canal of the untested ear being open)

$TL_{BSR(22)}$ Transmission loss (transtemporal attenuation) of the test signal (generated by the receiver R_2) in passing through the temporal bone to the cochlea of the untested ear

$TL_{BNR(22)}$ Transmission loss (transtemporal attenuation) of the masking noise (generated by the receiver R_2) in passing through the temporal bone to the cochlea of the untested ear

$TL_{BNR(21)}$ Transmission loss (transcranial attenuation) of the masking noise (generated by the receiver R_2) in passing through the skull to the cochlea of the tested ear (the auditory canal of the ear under test being open)

$TL_{BSV(F1)}$ Transmission loss of the test signal (generated by the vibrator V when it is placed on the forehead) in passing through the skin and the skull to the cochlea of the ear under test (the auditory canal of the ear under test being open)

$TL_{BSV(F2)}$ Transmission loss of the test signal (generated by the vibrator V when it is placed on the forehead) in passing through the skin and the skull to the cochlea of the untested ear (the auditory canal of the untested ear being open)

$TL_{ASV(F1)}$ Transmission loss of the test signal (radiated by the vibrator V when it is placed on the forehead) in passing through the air (air and/or metal head band) to the cochlea of the tested ear (normal middle ear and the auditory canal of the ear under test being open)

$TL_{ASV(F2)}$ Transmission loss of the test signal (radiated by the vibrator V when it is placed on the forehead) in passing through the air (air and/or metal head band) to the cochlea of the untested ear (normal middle ear and the auditory canal of the untested ear being open)

$TL_{BSV(M11)}$ Transmission loss of the test signal (generated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the skin and the temporal bone to the cochlea of the tested ear (the auditory canal of the ear under test being open)

$TL_{BSV(M12)}$ Transmission loss of the test signal (generated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the skin and the skull to the cochlea of the untested ear (the auditory canal of the untested ear being open)

$TL_{BSV(M12\ M11)}$ Difference between the transmission losses $TL_{BSV(M12)}$ and $TL_{BSV(M11)}$
$TL_{BSV(M12\ M11)} = TL_{BSV(M12)} \quad TL_{BSV(M11)}$

$TL_{BSV(F1\ M11)}$ Difference between the transmission losses $TL_{BSV(F1)}$ and $TL_{BSV(M11)}$
$TL_{BSV(F1\ M11)} = TL_{BSV(F1)} - TL_{BSV(M11)}$

$TL_{ASV(M11)}$

and the auditory canal of the ear under test being open)

$TL_{ASV(M12)}$ Transmission loss of the test signal (radiated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the air (air and/or metal headband) to the cochlea of the tested ear (normal middle ear and the auditory canal of the ear under test being open)

$TL_{BNV(F1)}$ Transmission loss of the masking noise (generated by the vibrator V when it is placed on the forehead) in passing through the skin and the skull to the cochlea of the tested ear (the auditory canal of the ear under test being open)

$TL_{BNV(F2)}$ Transmission loss of the masking noise (generated by the vibrator V when it is placed on the forehead) in passing through the skin and the skull to the cochlea of the untested ear (the auditory canal of the untested ear being open)

$TL_{ANV(F1)}$ Transmission loss of the masking noise (radiated by the vibrator V when it is placed on the forehead) in passing through the air (air and/or metal head band) to the cochlea of the tested ear (normal middle ear and the auditory canal of the ear under test being open)

$TL_{ANV(F2)}$ Transmission loss of the masking noise (radiated by the vibrator V when it is placed on the forehead) in passing through the air (air and/or metal headband) to the cochlea of the untested ear (normal middle ear and the auditory canal of the untested ear being open)

$TL_{BNV(M11)}$ Transmission loss of the masking noise (generated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the skin and the temporal bone to the cochlea of the tested ear (the auditory canal of the ear under test being open)

$TL_{BNV(M12)}$ Transmission loss of the masking noise (generated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the skin and the skull to the cochlea of the untested ear (the auditory canal of the untested ear being open)

$TL_{BNV(M12\ M11)}$ Difference between the transmission losses $TL_{BNV(M12)}$ and $TL_{BNV(M11)}$
$TL_{BNV(M12\ M11)} = TL_{BNV(M12)} - TL_{BNV(M11)}$

$TL_{BNV(F1\ M11)}$ Difference between the transmission losses $TL_{BNV(F1)}$ and $TL_{BNV(M11)}$
$TL_{BNV(F1\ M11)} = TL_{BNV(F1)} - TL_{BNV(M11)}$

$TL_{AN(M11)}$ Transmission loss of the masking noise (radiated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the air (air and/or metal headband) to the cochlea of the tested ear (normal middle ear and the auditory canal of the ear under test being open)

$TL_{AN(M12)}$ Transmission loss of the masking noise (radiated by the vibrator V when it is placed on the mastoid of the ear under test) in passing through the air (air and/or metal headband) to the cochlea of the untested ear (normal middle ear and the auditory canal of the untested ear being open)

ACKNOWLEDGMENT

I should like to express my deepest gratitude to Professor E Luscher, Director of the University Clinic of Oto Rhino Laryngology, Basel, for his constant encouragement during the preparation of the manuscript and to Professor Hallowell Davis, Director of Research, Central Institute for the Deaf, St Louis U S A , for his generous aid in correcting the Appendix "Explanation of the symbols"

REFERENCES

1 *American Standard Association spec Z*, 1938 24/244
2. AUBRY, M, 1945 L importance clinique de la conduction osseuse *Sem Hôp. Paris*, 12, 28 mars
3 AUBRY, M, CAUSSE, J, and CHAVANNE, L, 1938 Contribution a l etude de l'audiometrie *Ann Oto-laryng* (Paris), 8, 689–714
4 AUBRY, M, and GIRAUD, J CH, 1939 Le probleme de l assourdissement dans l'examen de l au
5 AUBR
1955
Internat Congress of Audiology, Paris, p 5–71
6 BARÁNY, E, 1938 A contribution to the physiology of bone conduction *Acta Oto-laryng* (Stockh) Suppl 26
7 BEKESY, G v, 1932 Zur Theorie des Horens bei der Schallaufnahme durch Knochenleitung *Ann Physik*, 13, 111 136
8 BÉKESY, G v, 1939 Über die piezoelektrische Messung der absoluten Horschwelle bei Knochenleitung *Akust Z*, 4, 113–125
9 BEKESY, G v, 1941 Über die Schallausbreitung bei Knochenleitung *Z Hals, Nas u. Ohrenheilk*, 47, 430–442.
10 BEKESY, G v, 1948 Vibration of the head in a sound field and its role in hearing by bone conduction *J Acoust Soc Amer*, 20, 749–760
11 BÉKESY, G v, 1949 The structure of the middle ear and the hearing of one's own voice by bone conduction *J Acoust Soc Amer* 21, 217–232
12 BEKESY, G v, 1960 *Experiments in hearing* McGraw Hill, New York, Chap 6
13 BEKESY G v, and ROSENBLITH, W A, 1951 The mechanical properties of the ear In STEVENS, S S *Handbook of experimental psychology* John Wiley, New York, Chap 27, 1075–1115
14 BEZOLD, F, 1897 *Über die funktionelle Prüfung des menschlichen Gehororgans* J F Bergmann Wiesbaden
15 BEZOLD, F, 1903 Die Horprufung mit Stimmgabeln bei einseitiger Taubheit und die Schlusse, welche sich daraus fur die Knochenleitung und die Funktion des Schalleitungs apparates ziehen lassen *Z Ohrenheilk*, 45, 262–274
16 BILGER, R C, and HIRSH I J 1956 Masking of tones by bands of noise *J Acoust Soc Amer*, 28, 623–630
17 BUNCH, C C, 1943 *Clinical audiometry* C V Mosby Comp., St Louis, p 54–55
18 BURGEMEESTRE, A J 1953 Auditory masking in continuous audiometry *Acta Oto-laryng* (Stockh), 43, 506–516
19 BURGEMEESTRE, A J 1953 Auditory masking in continuous audiometry *Proc First Internat Congress of Audiology* Leiden p 77–80
20 CARHART, R 1950 Clinical application of bone conduction audiometry *Arch Otolaryng*, 51 798–808
21 CARHART, R, 1958 Audiometry in diagnosis *Laryngoscope* (St Louis), 68, 253–277
22 CARLISLE, R W PEARSON, H A and WERNER P R, 1947 Construction and calibration of an improved bone conduction receiver for audiometry *J Acoust Soc Amer*, 19, 632–638
23 CHOCHOLLE, R 1959 A propos des seuils de conduction osseuse en presence de differences de sensibilite importantes entre les deux oreilles *Ann Oto laryng* (Paris), 71, 76–81
24 CLAUS G 1926 Über Kopfknochenleitung und gekreuzte Perzeption. *Mschr Ohrenheilk*, 60 316–328
25 DEAN C E, 1930 Audition by bone conduction *J Acoust Soc Amer*, 2, 281–296
26 DENES, P and NAUNTON R F 1952 Masking in pure tone audiometry *Proc Roy Soc Med*, 45, 790–794

27. EGAN, J P, and HAKE, H W, 1950 On the masking pattern of a simple auditory stimulus *J Acoust Soc Amer*, *22*, 622–630
28 EHMER, R H, 1959 Masking patterns of tones *J Acoust Soc Amer*, *31*, 1115–1120
29 EHMER, R H, 1959 Masking by tones vs noise bands *J Acoust Soc Amer*, *31*, 1253–1256
30 EVERBERG, G, 1953 Studies on the bone conduction in occlusion of the meatus and in chronic sound conduction deafness *Acta Otolaryng* (Stockh), *13*, 517–525
31 FELDTKELLER, R, and ZWICKER, E, 1956 *Das Ohr als Nachrichtenempfanger* S Hirzel, Leipzig
32 FLETCHER, H, 1940 Auditory patterns *Rev mod Phys*, *12*, 47–65
33 FOURNIER, J E, 1953 Sur le mécanisme de la conduction osseuse I Les phénomènes principaux et les théories de la conduction osseuse *Ann Oto laryng* (Paris), *70*, 401–433
34 FOURNIER, J E, 1953 Sur le mécanisme de la conduction osseuse II Complément à la théorie de la conduction osseuse, le problème de l'assourdissement *Ann Oto laryng* (Paris), *70*, 527–543
35 FOURNIER, J E, 1954 The false Bing" phenomenon Some remarks on the theory of bone conduction *Laryngoscope* (St Louis), *64*, 29–34
36 FOURNIER, J E, 1959 Sur les procédés directs de mesure des seuils de conduction osseuse sans élimination de l oreille contralatérale *Ann Oto laryng* (Paris), *76*, 68–75
37 FOURNIER, J E, 1959 Bone conduction In KOBRAK, H G *The middle ear* The University of Chicago Press, Chicago, Chap 5
38 FOURNIER, J E, and RAINVILLE, M J, 1954 Der «Faux Bing» Test und seine klinische Bedeutung *Z Laryng Rhinol*, *33*, 481–485
39 FOURNIER, J E, and RAINVILLE, M J, 1954 L intérêt clinique du phénomène du «Faux Bing», une nouvelle épreuve audiométrique *Ann Oto laryng* (Paris), *71*, 582–587
40 FOWLER, E P SR, 1925 Fundamentals of bone conduction *Arch Otolaryng*, *2*, 529–542
41 FOWLER, E P, 1951 Signs, emblems and symbols of choice in plotting threshold audiograms *Arch Otolaryng*, *53*, 129 133
42 FRANKE, E K, v GIERKE, H E, GROSSMAN, F M, and v MITTERN, W W, 1952 Jaw motions relative to the skull and their influence on hearing by bone conduction *J Acoust Soc Amer*, *24*, 142–146
43 GALAMBOS, R, 1956 Suppression of auditory nerve activity by stimulation of efferent fibers to cochlea *J Neurophysiol*, *19* 424–437
44 GOODHILL, V, and HOLCOMB, A L, 1955 Cochlear potentials in the evaluation of bone conduction *Ann Otol* (St Louis), *64* 1213 1233
45 GREENWOOD, D D 1961 Auditory masking and the critical band *J Acoust Soc Amer*, *33* 484–502
46 GROEN J J, 1953 Audiometers Criteria and design *Proc First Internat Congress of Audiology*, Leiden, p 24–26
47 GROEN J J, and HELLEMA, A C M 1960 Binaural speech audiometry *Acta Otolaryng* (Stockh), *52*, 397–414
48 GUILD S R 1936 Hearing by bone conduction the pathways of transmission of sound *Ann Otol* (St Louis), *45*, 736–768
49 HARBERT F, 1957 Masking levels for clinical use *Arch Otolaryng*, *66*, 214–222
50 HARBERT F 1958 The clinical masking level *Ann Otol* (St Louis) *67* 332–340
51 HARBERT F and SATALOFF, J A, 1955 Clinical applications of recruitment and masking *Laryngoscope* (St Louis), *65* 113 123
52 HARRIS J D HAINES, H L, and MYERS, C K 1953 A helmet held bone conduction vibrator *Laryngoscope* (St Louis) *63*, 998 1007
53 HART, C W and NAUNTON R F, 1961 Frontal bone conduction tests in clinical audiometry *Laryngoscope* (St Louis), *71* 24–29
54 HAWKINS, J E and STEVENS, S S 1950 The masking of pure tones and of speech by white noise *J Acoust Soc Amer*, *22*, 6–13
55 HERZOG H 1926 Das Knochenleitungsproblem Theoretische Erwägungen *Z Hals, Nas u Ohrenheilk* *15* 300 306
56 HIRSH I, 1952 *The measurement of hearing* McGraw Hill, New York

57 Hood, J D, 1957 The principles and practice of bone conduction audiometry A review of the present position *Proc Roy Soc Med* 50 689–697
– 1960 The principles and practice of bone conduction audiometry A review of the present position *Laryngoscope* (St Louis), *70*, 1211–1228
58 Hughson, W and Westlake, H, 1944 Manual for program outline for rehabilitation of aural casualties both military and civilian *Trans Amer Acad Ophthal Otolaryng*, Suppl 49, 1–15
59 Huizing, E H, 1960 Bone conduction The influence of the middle ear *Acta Otolaryng* (Stockh), Suppl 155
60 Jerger, J F, and Tillman, T W 1960 A new method for the clinical determination of sensorineural acuity level (SAL) *Arch Otolaryng*, *71* 948 955
61 Jerger, J F, Tillman, T W, and Peterson J L 1960 Masking by octave bands of noise in normal and impaired ears *J Acoust Soc Amer* *32* 385–390
62 Jukers A L, 1948 Observations on bone conduction in fenestration cases *Ann Otol* (St Louis) *57*, 28–40
63 Kelley N H, and Reger S N, 1937 The effect of binaural occlusion of the external auditory meati on the sensitivity of the normal ear for bone conducted sound *J Exp Psychol*, *21*, 211–217
64 Kietz, H 1950 Die Vertaubung bei der Horschwellenmessung mit Knochenschall *Arch Ohr, Nas, u Kehlk Heilk*, *157*, 125–135
65 Kietz, H, 1954 Die Vertaubung des abgewandten Ohres *Z Laryng Rhinol*, *33* 485–493
66 Kietz, H, 1956 Untersuchungen und Ergebnisse bei der Vertaubung des abgewandten Ohres *Arch Ohr, Nas u Kehlk Heilk* *169* 388–393
67 Kirikae, L, 1959 An experimental study on the fundamental mechanism of bone conduction *Acta Oto-laryng* (Stockh) Suppl 145
68 Kodman, F 1958 A method for verifying monaural deafness *Acta Otolaryng* (Stockh), *49*, 527–530
69 Konig, E, 1955 Les variations de la conduction osseuse en fonction de la force de pression exercee sur le vibrateur *Acustica* *5*, 234 243
70 Konig, E 1955 Les variations de la conduction osseuse en fonction de la force de pression exercee sur le vibrateur *Proc Second Internat Congress of Audiology* Paris, p 87–97
– 1957 Translation of the Beltone Institute for Hearing Research, 6, 1–12
71 Krainz W 1926 Das Knochenleitungsproblem Experimentelle Ergebnisse *Z Hals Nas u Ohrenheilk* *15* 306–313
72 Lane C E, 1925 Binaural beats *Phys Rev* *26* 401–412
73 Langenbeck B 1952 *Leitfaden der praktischen Audiometrie* G Thieme, Stuttgart
74 Langenbeck B 1954 Das Überhoren *Acta Otolaryng* (Stockh), *44* 462–470
75 Langenbeck B 1954 Das Überhoren und die richtige Vertaubung des besseren Ohres *Z Laryng Rhinol*, *33* 527–546
76 Langenbeck B 1956 *Leitfaden der praktischen Audiometrie* G Thieme Stuttgart
77 Langenbeck B 1958 Die Lateralisation des Knochenleitungshorens beim Weberschen Versuch *Arch Ohr Nas u Kehlk Heilk* *172* 451–456
78 Langenbeck B 1959 Die Lateralisation des Knochenleitungstones von Scheitel und Stirn (Weberscher Versuch) *Acta Otolaryng* (Stockh) *50* 406–410
79 Legouix J P and Tarab S 1959 Experimental study of bone conduction in ears with mechanical impairment of the ossicles *J Acoust Soc Amer* *31*, 1453–1457
80 Licklider J C R 1951 Basic correlates of the auditory stimulus In Stevens, S S *Handbook of Experimental Psychology* John Wiley New York Chap 25
81 Lidén G 1954 Speech audiometry An experimental and clinical study with swedish language material *Acta Otolaryng* (Stockh) Suppl 114
82 Lidén G Nilsson G and Anderson H 1959 Narrow band masking with white noise *Acta Otolaryng* (Stockh) *50* 116–124
83 Lidén G Nilsson G and Anderson H 1959 Masking in clinical audiometry *Acta Otolaryng* (Stockh) *50* 125–136
84 Lierle, M D and Reger S N 1946 Correlations between bone and air conduction acuity measurements over wide frequency ranges in different types of hearing impairments *Laryngoscope* (St Louis) *56* 187 224

85 LIGHTFOOT, C, 1960 The M R test of bone conduction hearing *Laryngoscope* (St Louis), *70*, 1552–1559

86 LINK, R, and ZWISLOCKI, J, 1951 Audiometrische Knochenleitungsuntersuchungen *Arch Ohr, Nas, u Kehlk Heilk*, *160*, 347–357

87 LITTLER T S, KNIGHT, J J, and STRANGE, P H, 1952 Hearing by bone conduction and the use of bone conduction hearing aids *Proc Roy Soc Med*, *45*, 783–790

88 LUCAE, A, 1864 Untersuchungen uber die sogenannte «Knochenleitung» und deren Verhaltnis zur Schallfortpflanzung durch die Luft, im gesunden und kranken Zustande. *Arch Ohrenheilk*, *1*, 303–352

89 LUSCHER, E, 1954 Vorschlage fur die vorlaufige Standardisierung der zu Diagnosezwecken verwendeten Reintonaudiometer *Z Laryng Rhinol*, *33*, 1–18

90 LUSCHER, E, 1954 Maskierung mit abgestimmtem Gerausch In ZOLLNER *Audiologie* Georg Thieme, Stuttgart, p 69

91 LUSCHER, E, and KONIG, E, 1954 Les problemes audiometriques de la conduction osseuse et de l assourdissement *Actas del primer congresso extraordinario de la sociedad internacional de audiologia* Buenos Aires, 1, 1956

92 LUSCHER, E, and KONIG, E, 1955 Die Vertaubung des Gegenohres bei der audiometrischen Bestimmung der Knochenleitung *Arch Ohr, Nas, u Kehlk Heilk*, *168*, 68–88

93 MARKLE, D M, FOWLER, E P, JR, and MOULONGUET, H, 1952 The audiometer Weber test as a means of determining the need for, and type of, masking *Ann Otol*, (St Louis), *61*, 888 900

94 MATZKER, J, 1954 Eine Methode zur genauen Messung des endokraniellen Heruberhorens beim Normalhorenden *Z Laryng Rhinol*, *33*, 619 625

95 MCCONNEL, F, and CARHART, R, 1952 Influence of fenestration surgery on bone conduction measurements *Laryngoscope* (St Louis), *62*, 1267–1292

96 MEISTER, F J, 1952 Physikalische Fragen und Gesichtspunkte bei der diagnostischen Gerausch Verdeckungsmessung am Ohr *Acustica* Suppl 2, 49–58

97 MEISTER, F J, 1954 *Akustische Meßtechnik der Gehorprufung* G Braun, Karlsruhe

98 MILLER, M H 1959 Transmission loss across the skull in a patient with known total monaural deafness *Laryngoscope* (St Louis) *69*, 100–102

99 NAUNTON, R F, 1957 Clinical bone conduction audiometry The use of a frontally applied bone conduction receiver and the importance of the occlusion effect in clinical bone conduction audiometry *Arch Otolaryng*, *66* 281–298

100 NAUNTON R F and FERNÁNDEZ C 1961 Prolonged bone conduction Observations on man and animals *Laryngoscope* (St Louis) *71* 306–318

101 NILSSON, G 1942 Some aspects of the differential diagnosis of obstructive and neural deafness *Acta Otolaryng* (Stockh) *30* 125 137

102 OEKEN, F W and MOBIUS, M 1961 Erfahrungen bei 76 Fußplattenplastiken an der Leipziger Universitats Hals Nasen Ohrenklinik *HNO Wegweiser*, *9*, 316–320

103 ONCHI Y 1954 The blocked bone conduction test for differential diagnosis *Ann Otol* (St Louis), *63*, 81 96

104 PALVA, T 1954 Masking in audiometry With special reference to the non thermal type of noise *Acta Otolaryng* (Stockh), Suppl 118, 156–172

105 PALVA T 1958 Masking in audiometry Further studies *Acta Otolaryng* (Stockh), *49* 229–239

106 PALVA T and OJALA L 1955 Middle ear conduction deafness and bone conduction *Acta Otolaryng* (Stockh), *45* 135–152

107 PAROW E 1957 Theorie und Praxis der Vertaubung *Arch Ohr Nas, u Kehlk Heilk*, *170*, 527–542

108 PETERSON J L 1959 *Masking by narrow bands of noise in various types of hearing loss* Unpublished doctoral dissertation Northwestern University

109 POHLMAN A G and KRANZ F W, 1925 The problem of middle ear mechanics Chapt III Binaural acuity for air and bone transmitted sound under varying conditions in the external auditory canal *Ann Otol* (St Louis) *34* 1224 1248

110 POHLMAN A G and KRANZ F W 1926 The influence of partial and complete occlusion of the external auditory canals on air and bone transmitted sound *Ann Otol* (St Louis), *35* 113–121

111 POHLMAN, A G, and KRANZ F W, 1926 Monaural tests on bone acuity under various conditions with some general comments on bone acuity *Ann Otol* (St Louis), 35, 632 641

112 POLITZER, A, 1908 *Lehrbuch der Ohrenheilkunde fur praktische Arzte und Studierende* F Enke, Stuttgart

113 RAINVILLE, M J, 1955 Nouvelle methode d assourdissement pour le releve des courbes de conduction osseuse *J Franç d'O R L*, *4* 851–858

1959 Translations of the Beltone Institute for Hearing Research, No 11

114 RASMUSSEN, G F, 1946 The olivary peduncle and other fiber projections of the superior olivary complex *J Comp Neurol*, *84* 141–219

115 RUF, C, 1932 Untersuchungen uber die Schalleitung im Schadel *Z Hals, Nas, u Ohrenheilk*, *31*, 356–369

116 RYTZNER C, 1954 Sound transmission in clinical otosclerosis *Acta Otolaryng* (Stockh), Suppl 117

117 SALTZMAN M 1949 *Clinical audiology* Grune & Stratton Inc, New York, p 6 and 55

118 SALTZMAN, M and ERSNER, M S, 1950 Masking and shadow hearing in bone conduction *Arch Otolaryng* 51 809–813

119 SCHAFER, T H, GALES R S, SHEWMAKER, C A, and THOMPSON, P O, 1950 The frequency selectivity of the ear as determined by masking experiments. *J Acoust Soc Amer*, *22*, 490–496

120 SCHMIDT, P H., and SPOOR, A, 1957 The place of an oscillator on the skull and the lateralisation of the sound *Pract Oto-rhino laryng* (Basel) *19*, 335–336

121 SCHUBERT, K, 1949 Auf Wegen zu neuen Horprufgeraten *Arch Ohr, Nas, u Kehlk Heilk*, *155*, 656–666

122 SCHUBERT, K., 1950 Der klinische Wert verschiedener audiometrischer Methoden *Arch Ohr, Nas, u Kehlk Heilk*, *157*, 328–340

123 SCHUBERT, K., 1951 Ein neues Audiometer und die damit erzielten Ergebnisse *Z Laryng Rhinol*, *30*, 11–26

124 SCHUBERT K., 1951 Vergleich alter und neuer Horprufmethoden *Arch Ohr, Nas u Kehlk Heilk*, *157*, 99–116

125 SIEGENTHALER, B M COHEN A. and RODES D 1957 Additional data on bone conduction shift when the external auditory meatuses are occluded *Arch Otolaryng*, *66*, 223–227

126 SMALL, A M, 1959 Pure tone masking *J Acoust Soc Amer*, *31*, 1619–1625

127 SMITH, K R, 1943 Bone conduction during experimental fixation of the stapes *J Exp Psychol*, *33*, 96–107

128 SOHOEL T, 1958 Masking in pure tone audiometry Its use and its limitations *J Laryng*, *72*, 959 969

129 SPARREVOHN U R, 1946 Some audiometric investigation of monaurally deaf persons *Acta Otolaryng* (Stockh) *34* 1 10

130 SPOOR A SCHMIDT P H and VAN DISHOECK H A E., 1957 The vibration on the skull of a bone conduction receiver and the lateralisation of the sound impression *Acta Oto laryng* (Stockh) *48* 304–307

131 STEVENS S S and DAVIS H 1938 *Hearing* Its Psychology and Physiology Wiley & Sons New York

132 SULLIVAN J A GOTTLIEB C C and HODGES W E, 1947 Shift of bone conduction threshold on occlusion of the external ear canal *Laryngoscope* (St Louis) *57* 690–703

133 TANNER W P 1958 What is masking? *J Acoust Soc Amer* *30*, 919 921

134 TARAB S 1958 Le mecanisme de l epreuve de Weber et les variations auditives en fonction de la pression d air dans l oreille externe et moyenne *Rev Laryng* (Bordeaux), *79*, 1223–1259

135 TARAB, S 1959 Le mecanisme de l epreuve de Weber *Pract Oto rhino laryng* (Basel), *21* 9–14

136 *Tillman* T W 1959 *Masking by narrow bands of noise in normal and impaired ears* Unpublished doctoral dissertation Northwestern University

137 TSCHIASSNY K 1952 Mechanism of shadow hearing *Arch Otolaryng*, *55*, 22 30

138 URBANTSCHITSCH V 1877 Uber die von der Hohe des Stimmgabeltones und von der Applikationsstelle abhangige Schalleitung durch die Kopfknochen *Arch Ohrenheilk*, *12* 20 250

139 *Veteran's Administration Spec VA HM 956* (Revised 1944)
140 WANGEMANN, H, 1933 Beitrag zur Frage der Knochenleitung bei einseitiger Taubheit (Untersuchungen am Otoaudion) *Z Hals, Nas u Ohrenheilk*, *32*, 603–614
141 WATSON, N, and GALES R, 1943 Bone conduction threshold measurements, effects of occlusion, enclosures, and masking *J Acoust Soc Amer*, *14*, 207–215
142 WEGEL, R L, and LANE, C H, 1924 The auditory masking of one pure tone by another and its probable relation to the dynamics of the inner ear *Phys Rev*, *23*, 266–285
143 WEILLE, F L, and GARGANO, S, 1953 Facts and fallacies of bone conduction *Laryngoscope* (St Louis), *63*, 182–211
144 WEVER, E G, and LAWRENCE, M, 1954 *Physiological Acoustics* Princeton University Press, Princeton, New Jersey
145 WEVER, E G, Lawrence, M., and SMITH, K R, 1948 The effects of negative air pressure in the middle ear *Ann Otol* (St Louis), *57*, 418–428
146 ZWICKER, E, FLOTTORP, G, and STEVENS, S S, 1957 Critical band width in loudness summation *J Acoust Soc Amer* *29*, 548–557
147 ZWISLOCKI, J, 1949 Über die Lautstärkeempfindung bei Knochenleitung *Acta Otolaryng* (Stockh), *37*, 239–244
148 ZWISLOCKI, J, 1951 Eine verbesserte Vertäubungsmethode fur die Audiometrie *Acta Otolaryng* (Stockh), *39*, 338–356
149 ZWISLOCKI, J, 1953 Acoustic attenuation between the ears *J Acoust Soc Amer*, *25*, 752–759

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta

OTO-LARYNGOLOGICA

S U P P L E M E N T U M 181

PRIMARY MALIGNANT EPITHELIAL TUMOURS OF THE MAXILLARY SINUS

A Clinical Study

BY

GIOVANNI ROSSI, GIACOMO DEMICHELIS
and ENRICO CHERUBINI

ACTA OTO LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 181

PRIMARY MALIGNANT EPITHELIAL TUMOURS OF THE MAXILLARY SINUS

A CLINICAL STUDY

by

GIOVANNI ROSSI

GIACOMO DEMICHELIS ENRICO CHERUBINI

From the Ear Nose and Throat Department

(Head Professor F BRUNETTI)

University of Turin

b) *small cell carcinoma,* a particular type of epithelial carcinoma with the same histological features as small cell carcinoma of the tracheobronchial tree The existence of this type of tumour in otorhinolaryngological organs has already been reported by one of us (Rossi, Rossi and Cherubini, Rossi and Menzio) with reference to the nasal cavities and the pharyngotonsillar region

Since this type of epithelial tumour featured in two of our cases, it may be of some interest to describe its main histological characteristics and the way in which it differs from lymphoblastic or lymphocytic reticulosarcoma The neoplastic cells of small cell carcinoma display the characteristic tendency of neoplastic epithelial cells to cluster in groups, cords and tubules, there is very little stroma between them and parenchyma and stroma are clearly separated (Figure 4)

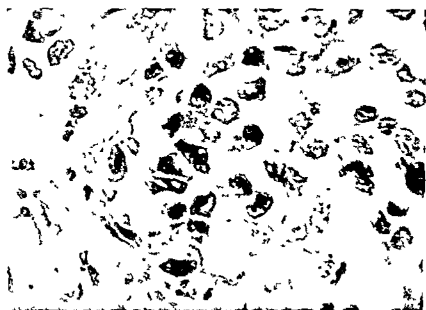


Fig 5 Case 22 — Small cell carcinoma High power view of Figure 4 showing clearly the features of the malignant cells (haematoxylin-eosin x 800)

In lymphoblastic or lymphocytic reticulosarcoma, as in all connective tissue tumours parenchyma and stroma are not clearly separated

The cells of this type of small cell carcinoma are larger than those of lympho blastic or lymphocytic reticulosarcoma (Figure 5)

Lymphoblastic and lymphocytic reticulosarcomas are deficient in argentophilic reticulum Useful pointers to differenential diagnosis are therefore not to be had from a study of the reticular component

he pes of carcinoma which featured in the present series are shown in Tab

TABLE 3

	Histological type	Case No	Total No of cases
	spindle-cell ca	4, 6, 8, 10, 12, 13 18, 19 23, 24, 33, 34	12
differentiated carcinomas	basal-cell ca	5, 9, 16, 21, 25, 26	6
	cylindrical-cell ca	17, 28, 30	3
	papilliferous ca	7, 14	2
	adenocarcinoma	11, 29	2
undifferentiated carcinomas	anaplastic ca	1, 2, 3, 15, 20, 27, 32	7
	small-cell ca	22, 31	2
A total of 25 differentiated carcinomas and 9 undifferentiated carcinomas			

SYMPTOMATOLOGY

1) *Initial symptoms*

The initial presenting symptoms of the patients of our series are listed in Table 4

TABLE 4

	Case No	Total No of cases
Cheek swelling	2 10 13	3
Epiphora	11 17	2
Epistaxis	6 27	2
Trismus	21 24	2
Rhinorrhoea	4 14	2
Respiratory stenosis	12	1
Facial neuralgia	3 7 8 9 19 26 33 34	8
Cutaneous paraesthesia	1 23 28 30 31	5
Cutaneous anaesthesia	32	1
Epistaxis with nasal obstruction	22 29	2
Epistaxis with rhinorrhoea	20	1
Epistaxis with cheek swelling	18	1
Respiratory stenosis with cheek swelling	15, 16	2
Rhinorrhoea with gingival swelling	5 25	2

Although standard radiography in axial projection does give some idea of the posterior extension of the tumour, axial transverse tomography provides a far clearer picture and makes it possible to assess the vertical spread of the tumour, as well as its relations with the posterior wall of the maxillary sinus and the pterygopalatine fossa

In other cases further radiographic investigation revealed bony erosion of the inferior part of the sinus In still further cases radiographic examination in standard projections was done after introducing a contrast medium into the maxillary sinus This was not done in all cases, but mainly in those with intrinsic tumours whose exact site had to be determined

In many cases radiographic examination with contrast medium does not provide particularly accurate information, owing to the constant presence of inflammatory processes of the sinus mucosa not yet directly involved in the neoplastic process



F ( ) — C cumscr bed intrinsic tumour affecting only the medial wall of the right
v e are no clinical signs of nvasion of the lateral wall of the nasal fossa

s i 18

Pre-operative biopsy was always done in cases where the neoplastic process had extended to areas accessible from the outside (25 cases)

In two cases histological examination was done during surgery while in a further 7 cases after the anterior wall of the maxillary sinus had been opened neoplastic masses were discovered whose microscopic features fully justified radical operation

In 9 cases in which pre operative biopsy was not possible histological examination of sinus washings was done

In 4 cases out of 9 malignant epithelial neoplastic cells were demonstrated beyond any doubt In a further 5 cases the findings were doubtful or negative

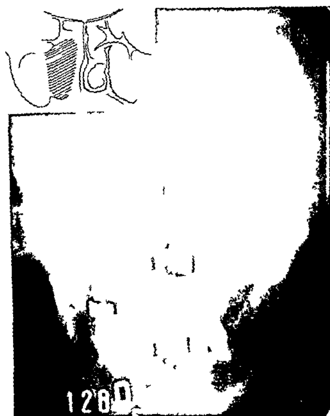


Fig Case 11 — Carcinoma [illegible] [illegible] medial tumour which originally arose in the medial wall of the right maxillary sinus and has invaded the lateral wall of the nasal fossa

ANATOMO CLINICAL CLASSIFICATION

The anatomo-clinical classification of malignant tumours of the maxillary sinus should not constitute an end in itself but should be based as far as possible, on data which can be used to assess prognosis in each case

In general by the time treatment is begun the tumour which arose originally in the maxillary sinus has already spread beyond the anatomical limits of the sinus itself to one or more adjacent anatomical structures Clinical, radiographic and tomographic findings as well as study of the operative specimen, generally provide sufficient evidence of this situation

Histological study of operative specimens has provided evidence that in cases in which the tumour appears to be circumscribed within the cavity of the maxillary



Fig 8 *Case 14* — Diffuse intrinsic tumour Radiography with contrast medium in the maxillary sinus The tumour involves the medial and inferior walls of the right maxillary sinus *The neighbouring anatomical structures have not been invaded*

On the strength of the foregoing observations the findings of clinical and radiographical examination and microscopic and gross anatomopathological evidence we have classified our series in two main groups each subdivided into two sub-groups

1) *Circumscribed tumours* that is primary tumours of the maxillary sinus which on commencement of treatment involve only one of the walls of the sinus Circumscribed tumours can be either



Fig 9 *Case 15* — Diffuse extrinsic superior tumour which originally arose in the left maxillary sinus and has invaded the ethmoid and orbit Tomography in transverse axial projection and operative finding show that the pterygopalatine fossa has not been invaded

a) *intrinsic* which neoplastic development takes place entirely within the sinus and invasion of bone structures if any is only demonstrable by histological examination (Figure 6) or

b) *extr* in which
skeleton of the maxillary sinus
or by studying the operative specimen it involves only one of the anatomical structures adjacent to the maxillar sinus

A further classification can be made according to site which may be

medial with expansion towards the nasal fossa (Figure 7)
inferior with expansion towards the hard palate and the alveolar process
posterior with expansion towards the pterygopalatine fossa
anterolateral with expansion towards the cheek
superior with expansion towards the ethmoid orbit, or malar bone

2) *Diffuse tumours* that is primary tumours of the maxillary sinus which on commencement of treatment involve two or more walls of this sinus As with the circumscribed tumours diffuse tumours can be sub-divided into two groups

a) *intrinsic tumours* in which the neoplasm develops entirely within the sinus, of which it involves two or more walls and invasion of bone structures, if any can only be demonstrated by histological examination (Figure 8)



b) *extrinsic tumours* in which the neoplasm has extended beyond the limits of the bony skeleton of the maxillary sinus and invaded two or more adjacent anatomical structures

A careful study of our material shows that within the diffuse tumour group a distinction can be made between neoplasms of *superior* exteriorization and those of *inferior* exteriorization For purposes of treatment and prognosis the lateral or medial outward development of the tumour has little importance Far more significant is the invasion of the pterygopalatine fossa which experience suggests can be reached even by tumours whose site of origin is the floor of the sinus

Thus, extrinsic diffuse tumours can be schematically represented as follows

diffuse superior extrinsic tumours	without invasion of the pterygopalatine fossa (Figure 9) with invasion of the pterygopalatine fossa
diffuse inferior extrinsic tumours	without invasion of the pterygopalatine fossa with invasion of the pterygopalatine fossa (Figure 10)

A complete classification on these lines is shown in Table 6

TABLE 6

				Case No	Total
Circumscribed tumours (14)	Intrinsic			23 32	2
	Extrinsic		medial	11, 17 27	3
			inferior	5	1
			superior	7 8 19 28 29	5
			posterior	21 24	2
			antero-lateral	10	1
Diffuse tumours (20)	Intrinsic			3 14	2
	Extrinsic	Superior	without invasion of the pterygopalatine fossa	12, 15 26	3
			with invasion of the pterygopalatine fossa	1 4 9 22 25 30 31	7
		Inferior	without invasion of the pterygopalatine fossa	2 13 33 34	4
			with invasion of the pterygopalatine fossa	6 16 18 20	4

METASTASES

1) *Lymph node metastases*

On commencement of treatment the existence of laterocervical lymph node metastases was verified in 6 out of the 34 cases comprising our series

In cases 9 19 21 and 25 there was enlargement of the superior carotid

jugular lymph node group on the same side as the tumour Microscopic examination revealed areas of necrosis in these lymph nodes This was subsequently verified by histological examination In cases 22 and 24, on the other hand, inspection of the carotid jugular region revealed a number of enlarged lymph nodes, in which histological examination alone demonstrated the presence of small groups of malignant cells

In cases 14 18, and 33 there also existed moderate hyperplasia of a number of superior lymph nodes of the carotid jugular chain on the same side as the

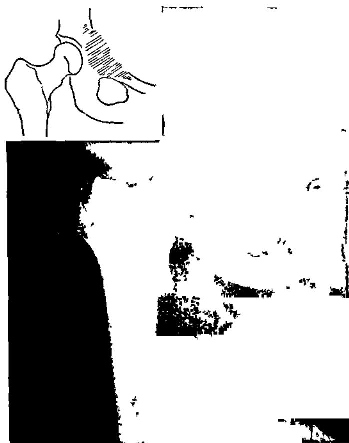


Fig 11 Case 22 — Metastases to the right ischium involving the anterior ramus of the pubis approximately 30 months after treatment

Laterocervical lymph node metastases were found in two cases of circumscribed extrinsic posterior tumour (case 21 and 24) in one case of circumscribed extrinsic tumour (case 19) and in three cases of diffuse extrinsic superior tumour (cases 9, 22, and 25) In 5 cases out of 6 (cases 9, 21, 22, 24 and 25) the tumour had invaded the pterygopalatine fossa

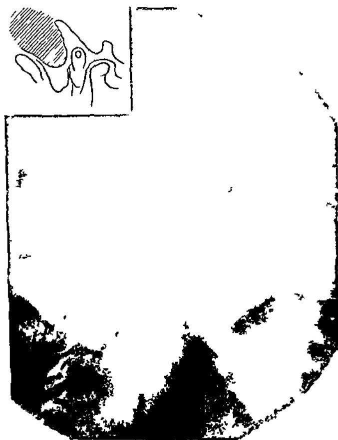


Fig 12 *Case 14* — Metastases to the right mastoid apophysis 6 years after treatment

With regard to histological type of tumour latero-cervical lymph node metastases were found in two cases of spindle cell carcinoma (cases 19 and 24) three cases of basal cell carcinoma (cases 9 21 and 25) and one case of small cell carcinoma (case 22)

2) *Distant metastases*

Systematic admission examination of all our patients revealed no distant metastases

In the period following treatment on the other hand distant metastases

appeared in a number of cases (cases 19 22 25 29 and 14) In three cases (cases 22 25 and 14) there were bone metastases of respectively the ischium (Figure 11) ribs and right mastoid apophysis (Figure 12) while in two cases (cases 19 and 29) the tumour had metastasized to the liver

In case 14 in which a mastoid metastasis appeared over six years from commencement of treatment the tumour subsequently metastasized to the lungs (Figure 13)

Distant metastases were found in one case of spindle cell carcinoma (case 19) one case of basal-cell carcinoma (case 25) one case of small-cell carcinoma (case 22) one case of adenocarcinoma (case 29) and one case of papilliferous carcinoma (case 14)



Fig 13 *Case 14* — Right pulmonary metastases observed 6 years 10 months after treatment

TREATMENT

Combined radiological and surgical treatment was carried out in all the cases of our series and consisted of

) re of the external carotid artery ipsilateral to the tumour and possible [illegible] te ocervical lymph nodes

b [illegible] umo r

c) r [illegible] therapy

d) ex [illegible] herap





Fig 14 *Case 34* — Diffuse extrinsic inferior tumour of the left maxillary sinus without invasion of the pterygopalatine fossa Radiography in frontal projection (A) and lateral projection (B) shows the position of the radium needles introduced into the operative cavity

None of the patients was submitted to pre operative roentgenotherapy, which we do not feel is justified in cases of malignant epithelial tumour of the maxillary sinus There is nearly always a concomitant suppurative process which always makes it very difficult to define the area to be irradiated and exposes the patient to the risk of radionecrosis

In all our patients surgery was carried out under general anaesthesia

Ligature of the external carotid artery ipsilateral to the tumour was performed in all the patients The advantages of opening the carotid jugular region are twofold on the one hand, it makes it possible to reduce intra operative haemor rhage and avoid postoperative haemorrhage, on the other, it facilitates direct exploration and possible removal of the laterocervical lymph nodes

Removal of the laterocervical lymph nodes was performed in 9 out of the 34 cases (case 9, 19, 21, 25, 22, 24, 14, 18 and 33) In all these cases there was clinical evidence of enlarged cervical lymph nodes

Histological examination showed that the tumour had metastasized to the lymph nodes in cases 9, 19, 21, 22 24 and 25, while in cases 14, 18 and 33, lymph node enlargement was due to reactive hyperplasia in the absence of metastatic invasion

In most cases classical paralateronasal and nasogenial incision was performed if necessary together with Zange's horizontal palpebral incision In one case (case 5) of circumscribed extrinsic inferior tumour, operation was performed through an Ohngren incision

Removal of the tumour was carried out by surgical destruction of the affected anatomical structures planned according to the site and mode of tumour spread

The principle underlying all these operations was to remove the tumour in healthy tissue For this reason, in circumscribed tumours, both intrinsic and extrinsic, resection was confined to the involved sinus walls and neighbouring anatomical structures due account being taken of the mode of tumour spread

Total resection of the superior maxilla was performed in all cases of diffuse tumour and completed, if necessary by resection of the malar bone exenteration of the orbit and ethmoidectomy

In all cases in which the tumour, whether circumscribed or diffuse involved the posterior wall of the maxillary sinus and in which there was thus the possibility of neoplastic invasion of the pterygopalatine fossa surgical removal was alway preceded by electrocoagulation of the tumour carried out with large coagulant electrodes connected to a suitable high tension unit Since coagulated neoplastic tissue i[illegible] a different colour from normal tissue its progression in the pterygopalatine fossa can [illegible] followed fairly accurately

Be[illegible]g to skin suture tubes of radium were inserted in the operati[illegible]e [illegible]ll cases we used 5 or 10 mg radium tubes with 1 mm Pt filtrat[illegible]er and position of the tubes being designed to provide homogeneo[illegible] the most likely area of neoplastic persistence, they

were maintained in place with a suitable pack and their position determined radiographically (Figures 14 A and B)

Dosage varied according to the size of area to be irradiated, although it never exceeded 20 to 30 mcd over a 3 7 day course This dose corresponds approximately to 1 mcd per cm^2 of area irradiated by an effective dose With these treatment factors no signs of radionecrosis in the adjacent bone structures were observed

Within 20 30 days of intracavitary radium therapy, treatment was completed with external roentgenotherapy in the Turin University Department of Radiology As high energy sources were not available at the time of the present investigation, all the patients were subjected to traditional roentgenotherapy, with a half value layer of not less than 1 5 mm Cu Energies of 180 200 kV with a minimum filtration of 1 mm Cu + 1 mm Al were used throughout Radiation beams were for the most part collimated with square collimators of side 4 5 cm The beams were suitably angled and transmitted through 3 4 ports, two of which (one anterior, the other lateral) were situated on the same side as the lesion, the others on the opposite side

A daily tumour dose of 100 150 r was administered the cumulative tumour dose amounting to not less than 3000 r

Within 2 3 months of completion of this first course of roentgenotherapy, all the patients were subjected to a second course employing the same treatment factors and providing a cumulative tumour dose of 2000 r

No irreversible cutaneous or mucosal reactions attributable to roentgenotherapy were observed

Apart from radiation treatment of the primary tumour, penetrating radiation (half value layer of not less than 1 5 mm Cu) was administered with direct fields varying between 8 x 10 cm and 12 x 12 cm on the cervical region of the 6 patients in whom the laterocervical metastatic lymph nodes had been removed (cases 9, 19, 21, 22 24 25) The surface dose for each field was in the region of 3000 r

GENERAL RESULTS

None of our patients died from postoperative complications Since our series consists of patients admitted up to 30th April 1957 evaluation of five year survival is possible

1) PATIENTS WHO DIED WITHIN 5 YEARS OF COMMENCEMENT OF TREATMENT

In Table 7 the survival times and causes of death of the patients who died within 5 years of commencement of treatment are shown

[illegible]

TABLE 7

No of deaths within	Case No	Causes of death
1 year (2)	8	Local recurrence and cachexia
	9	Local recurrence and cachexia
2 years (2)	10	Cardiocirculatory insufficiency
	21	Local recurrence and cachexia
3 years (8)	4	Myocardial infarction
	6	Local recurrence and cachexia
	19	Local recurrence and hepatic metastases
	22	Neoplastic cachexia with local recurrence and metastases to the ischium
	24	Cardiocirculatory insufficiency
	25	Metastases to the 4th and 5th left ribs with no signs of local recurrence
	26	Local recurrence and cachexia
	27	Local recurrence and cachexia
4 years (3)	2	Local recurrence and cachexia
	5	Local recurrence and cachexia
	30	Laterocervical lymph node metastases with no signs of local recurrence
5 years (1)	1	Local recurrence and cachexia

A study of Table 7 shows that 16 patients out of 34 died within 5 years of commencement of treatment Two died in the first year, 2 in the second year, 8 in the third year, 3 in the fourth year and 1 in the fifth year Three of these 16 patients (cases 10, 4 and 24) died in the second and third years, respectively, for causes unrelated to the neoplastic process, in cases 10 and 24 from cardiocirculatory insufficiency and in case 4 from myocardial infarction

In the other 13 patients the tumour was the direct cause of death

Neoplastic cachexia with local recurrence was the cause of death in 9 cases (cases 8 9 21 6 26 27 2 5 and 1), while in one case (case 19) local recurrence appeared simultaneously with hepatic metastases, in one patient (case 22) there was local recurrence with metastases to the iliac bone, while in a further two patients (cases 25 and 30) neoplastic lesions arose in the ribs and laterocervical lymph nodes respectively in the absence of clinical signs of local neoplastic recurrence

2 PATIE[illegible] ALIVE 5 YEARS AFTER COMMENCEMENT OF TREATMENT

In T b[illegible] data relating to the patients still living 5 years after commencement of t[illegible] t are shown

A study of Table 8 shows that 18 out of the 34 patients were still in good general condition 5 years after commencement of treatment. Of these 18 patients 5 died from causes unrelated to the neoplasm (cases 7, 20, 12, 17 and 15) after respectively, 6 years, 6 years, 7 years, 7 years, and 10 years from commencement of treatment.

In case 29, death 5 years 10 months following completion of treatment occurred as a result of secondary neoplastic cachexia and hepatic metastases with no signs of local neoplastic recurrence.

In case 14, 6 years 2 months following completion of treatment there appeared a right mastoid metastasis, which was submitted to surgery. Nine months later pulmonary metastatic diffusion appeared with no clinical evidence of local neoplastic recurrence.

TABLE 8

Patients still alive after	Case No	Remarks
	23	Alive and well
	29	Died after 5 years 10 months from hepatic metastases without signs of local neoplastic recurrence
5 years	31	Alive and well
(6)	32	Alive and well
	33	Alive and well
	34	Alive and well
	7	Died after 6 years 4 months from cerebral haemorrhage
6 years	14	Operated on for right mastoid metastasis. Died after 6 years 11 months from pulmonary metastasis
(4)	20	Died after 6 years and 5 months from myocardial infarction
	28	Alive and well
7 years	12	Died 7 years and 2 months from cerebral haemorrhage
(3)	16	Alive and well
	17	Died from cardiocirculatory insufficiency
8 years	11	Alive and well
(2)	18	Alive and well
9 years (1)	13	Alive and well
10 years (1)	15	Died after 10 years 3 months from cerebral haemorrhage
11 years (1)	3	Alive and well

a) Five-year survival in relation to histological features

Table 9 incorporates the findings in the 18 patients who survived over 5 years from commencement of treatment.

TABLE 9

	Histological type	Case No.	Total No. of cases
Differentiated carcinoma	spindle-cell ca.	8 12 13 18 33 34	6
	basal-cell ca.	16	1
	cylindrical-cell ca.	17 28	2
	[illegible] ca.	7 14	2
	adenocarcinoma	11 29	2
Undifferentiated carcinoma	anaplastic ca.	3 15 20 32	4
	small-cell ca.	31	1

Comparing Table 9 with Table 3, it is seen that five-year survival was attained in half the patients suffering from spindle-cell carcinoma (6 cases out of 12), while only one of the 6 cases of basal-cell carcinoma attained this survival time.

The 2 cases of cylindrical-cell carcinoma and the 2 cases of adenocarcinoma both survived the five-year limit, which was reached by 2 of the 3 cases of cylindrical-cell carcinoma.

Four of the 7 cases of anaplastic carcinoma and one of the 2 cases of small-cell carcinoma were still in good general condition with no signs of local recurrence 5 years from commencement of treatment.

Summing up the foregoing data, we see that five-year survival was achieved in 13 of the 25 cases of differentiated carcinoma and in 5 of the 9 cases of undifferentiated carcinoma.

b) Five-year survival in relation to interval between onset of the symptoms and commencement of treatment

The data concerning five-year survival in relation to the interval between onset of symptomatology and commencement of treatment are incorporated in Table 10.

From Table 10 it is seen that in 12 of the 18 patients who survived over 5 years, treatment had been begun within 6 months of the onset of symptoms.

In the other 6 cases treatment had been begun after, respectively, 8, 9, 10, 11, [illegible] and 24 months.

[illegible] mpare Table 10 with Table 5, it is seen that of the 24 cases in which [illegible] was begun within 6 months of onset of symptoms, 6 survived [illegible] in good general condition, with no signs of local neoplastic [illegible]

re [illegible]

lera [illegible] ISI

TABLE 10

		Case No (alive after 5 years)	Total No of cases
Interval between onset of symptoms and commencement of treatment	1 month	31	1
	2 months	3, 14	2
	3 months	13 16 32 34	4
	4 months	11, 15, 29	3
	5 months	33	1
	6 months	23	1
	8 months	12	1
	9 months	28	1
	10 months	20	1
	12 months	7	1
	18 months	17	1
	24 months	18	1



Fig. 15 *Case 11* — Right circumscribed extrinsic medial tumour (ade[illegible] patient 8 years after treatment

The findings obtained from the present investigation do not show a greater five-year survival rate in patients in whom treatment had been begun within 6 months of onset of symptoms than in those in whom treatment had been begun after 6 months from onset of symptoms.

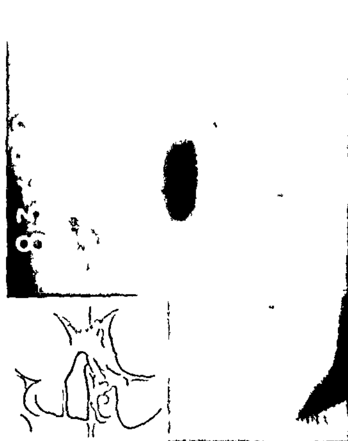


Fig. 10. Case 11. — Right circumscribed extrinsic medial tumour (ad scaenomeum). Radiographic examination 5 years after treatment. Compare this figure with the radiograph shown in Fig. 7 which was taken before treatment.

c) *Five-year survival in relation to anatomoclinical features*

By comparing Table 5 with Table 6 it is seen that the 18 cases in which a five-year survival was reached include 7 of circumscribed tumour (cases 7, 11, 17, [illegible] 32) and 11 of diffuse tumour (cases 3, 14, 12, 15, 31, 13, 33, 34 [illegible]

[illegible] tumour (cases 23 and 32), circumscribed tumour, cases [illegible] five years.

[illegible] tumours, 2 of the 3 cases of medial



Fig [illegible] Case 16 — Left diffuse extrinsic inferior tumour (basal-cell carcinoma) Radiograph taken before treatment The tumour has invaded the nasal fossa and alveolo-palatine region



Fig [illegible] Case [illegible] — The tumour was removed [illegible]

[illegible]

tumour (cases 11 and 17) (Figs 15 and 16) and 3 of the 5 cases of superior tumour (cases 7 28 and 20) survived over five years

In the 2 cases of extrinsic superior tumour in which exenteration of the orbit had been done (cases 8 and 19) the patient died shortly afterwards from local neoplastic recurrence with cachexia



Fig 19 *Case 16* — Radiography 7 years after treatment

In the group of diffuse extrinsic superior tumours only 3 out of the 10 patients survived over five years (cases 12 15 and 31) while in the group of diffuse extrinsic inferior tumours 6 of the 8 patients survived over five years (cases 13 33, 34 20 16 and 18) Figs 17 18 19 and 20)

With egard to the 13 cases involving invasion of the pterygopalatine fossa circumscribed trinsic posterior tumour in cases 30 31 22 25 1 9 and 4 diffuse extri s ior tumour in cases 6 20 16 and 18) only 4 patients (cases 31 13 33 a survived over five years Only one (case 31) of the 7 cases of

extrinsic superior tumour with invasion of the pterygopalatine fossa survived over five years while 3 of the 4 patients of extrinsic inferior tumour with invasion of the pterygopalatine fossa (cases 13 33 and 34) were alive and well at the end of the five year period

Among the patients with diffuse extrinsic tumours without invasion of the pterygopalatine fossa 2 of the 3 cases with superior outward expansion (cases 12 and 15) and 3 of the 4 cases with inferior tumour expansion (cases 20 16 and 18) survived over five years



Fig 20 *Case 16* — The patient years after treatment

d) *Five year survival in relation to lymph node metastase*

On commencement of treatment cases 9 19 21 22 24 and 25 [illegible] metastases of the upper group of the carotid jugular lymph nodes on [illegible] side

In cases 9 19 21 and 25 these metastases were verified cl[illegible] logically while in cases 22 and 24 the presence of malignant cells was [illegible] by histological examination alone None of the 6 patients survived [illegible]

In cases 21 and 9 the patients died from local recurrence [illegible]

cachexia, within the first and second years, respectively, from commencement of treatment The patient of case 19 died within the third year from local neoplastic recurrence with hepatic metastases In case 22 the patient also died within the third year from neoplastic cachexia with metastases to the ischium while the patient of case 25 died within the third year from costal metastases with no signs of local recurrence

In case 24 the patient died within the third year from cardiocirculatory insufficiency and not from causes related to the maxillary sinus tumour

CONCLUDING REMARKS

It is the object of the present paper to present the results obtained from the combined radiological and surgical treatment of *primary malignant epithelial tumours of the maxillary sinus*

The series reported consists of patients admitted to the Turin University Ear Nose and Throat Department between 1st May 1951 and 30 April 1957 All 34 patients were suffering from primary malignant epithelial tumours of the maxillary sinus which had not been previously subjected to any form of treatment

Detailed information regarding the postoperative course and long term results was available in all cases

From a study of the series presented it is possible to draw the following conclusions

1) There is a marked predominance of males (23 cases out of 34) with a male/female ratio of 2 1

2) The series includes 4 patients below 40 years of age Three of these 4 patients were female Approximately two thirds of the cases (23 out of 34) concerned patients between 40 and 60 years of age

3) In 3 of the patients tumours featured in the family history The mothers *of these patients had died from gastric tumour*

4) In only one of the cases was there a history of trauma to the maxillary sinus in which a tumour subsequently developed

One patient was suffering from active syphilis A further 2 patients had a history of syphilitic infection although serum tests were negative on admission

5) Three patients had been operated on for chronic maxillary sinusitis ipsilateral to the tumour site A further 3 patients were found on admission to have apical granulomas of the premolars and molars corresponding to the subsequent tumour site in the maxillary sinus

6) From an anatomopathological point of view the series can be divided into 2 main groups the first comprising patients with differentiated carcinoma (25 cases) and the second those with undifferentiated carcinoma (9 cases) The first group includes 12 cases of spindle cell carcinoma 6 cases of basal-cell carcinoma,

3 cases of cylindrical cell carcinoma, 2 cases of papilliferous carcinoma and 2 cases of adenocarcinoma The second group comprises 7 cases of anaplastic carcinoma and 2 cases of small-cell carcinoma

7) Of particular note was the finding in the maxillary sinus of a small-cell carcinoma possessing the histological features of the small-cell carcinoma found in the tracheobronchial tree These tumours are generally thought to originate in the cells of the basal layer of the cylindrical lining epithelium of the mucosa

8) On the basis of the clinical and radiographic findings and on microscopic, surgical and gross anatomopathological evidence, our cases can be classed in 2 main groups circumscribed tumours and diffuse tumours, each of which can be further sub-divided into 2 sub-groups

Circumscribed tumours are tumours involving one of the walls of the maxillary sinus and developing entirely within the sinus (*circumscribed intrinsic tumours*)

Circumscribed tumours which have extended beyond the anatomical limits of the maxillary sinus and invaded one of the adjacent anatomical structures may be classed as *circumscribed extrinsic tumours* (*medial, inferior, superior, posterior and anterolateral*)

Diffuse tumours include all primary tumours of the maxillary sinus which on commencement of treatmente are found to involve two or more walls of the maxillary sinus When the neoplasm develops entirely within the sinus, it is termed a *diffuse intrinsic tumour* when it invades the adjacent anatomical structures it is termed a *diffuse extrinsic tumour*

9) A study of long term results suggests that a factor of paramount impor tance for prognosis is whether or not the tumour has invaded the pterygopalatine fossa

Evidence was also obtained that invasion of the pterygopalatine fossa can even occur with tumours originating in the antero-inferior half of the maxillary sinus

Our findings therefore justify a further subdivision of diffuse extrinsic tumours into *diffuse extrinsic superior tumours* and *diffuse extrinsic inferior tumours* the former involving the ethmoid, orbit and zygomatic bone and the latter the nasal fossa palate alveolar process and vestibule of the mouth For each of these two sub-groups attention was paid to whether or not the pterygopalatine fossa had been invaded by the neoplastic process

10) On the basis of the foregoing classification, the 34 cases of our series can be considered as follows

— Circumscribed intrinsic tumours (2),

— Circumscribed extrinsic tumours (12)

— Diffuse intrinsic tumours (12),

— Diffuse extrinsic superior tumours with invasion of the pterygopalatine fossa (7)

— Diffuse extrinsic superior tumours without invasion of the pterygopalatine fossa (3),

— Diffuse extrinsic inferior tumours with invasion of the pterygopalatine fossa (4),

— Diffuse extrinsic inferior tumours without invasion of the pterygopalatine fossa (4)

11) In 14 cases the initial presenting symptoms were neurological (neuralgia and cutaneous paraesthesias and anesthesia), in 8 cases rhinological (epistaxis rhinorrhoea and nasal obstruction) in 2 cases ocular (epiphora), and in 3 cases disfiguring

In 2 cases the first symptom of tumour was trismus, while in 5 cases the tumour manifested itself in rhinological symptoms associated with facial deformity

12) Most of the patients (24 out of 34) were admitted to the Clinic within 6 months of onset of symptomatology

13) In 6 of the 34 cases there were laterocervical lymph node metastases on commencement of treatment In 4 of these 6 cases laterocervical lymph node metastases were verified by clinical and histological examination while in 2 cases histological examination of a number of slightly enlarged lymph nodes alone provided sufficient evidence of metastatic malignant cells

In one case laterocervical lymph node metastases appeared 3 years following commencement of treatment with no signs of local neoplastic recurrence

14) The series included 5 cases of distant metastases 2 to the liver, 1 to the ischium, 1 to the ribs and 1 to the mastoid apophysis with subsequent metastasization to the lungs These metastases appeared in the period following treatment

15) With regard to diagnostic methods transverse axial tomography was found particularly useful it reveals the exact relationship between the tumour and the pterygopalatine fossa whose invasion entails an unfavourable prognosis

Standard radiography and frontal tomography provide useful information on the spread of the tumour towards the structures lying above below and medially to the maxillary sinus

The introduction of contrast medium into the maxillary sinus is of value only in cases of intrinsic tumour

16) Cytological examination of sinus washings was performed in the 9 cases in which pre operative biopsy was not possible In 4 of these cases malignant epithelial cells were demonstrated beyond any doubt In the other 5 cases the cytological findings were doubtful or negative

17) All the patients of the series underwent combined radiological and surgical treatment which included ligature of the external carotid and the possible removal of the lymph nodes excision of the maxillary tumour intracavitary radium therapy and external roentgenotherapy

None of the patients underwent pre-operative roentgenotherapy All were operated on under general anaesthesia. Ligature of the external carotid was performed in all cases, whereas removal of the laterocervical lymph nodes was only performed in the 9 cases in which there was clinical evidence of their enlargement.

18) In only one case was operation performed through an Ohngren incision. In all other cases the classical paralateronasal incision was used, associated if necessary with Zange's horizontal palpebral incision.

In circumscribed tumours, both intrinsic and extrinsic, resection was confined to the sinus walls affected by the tumour and the neighbouring anatomical structures

In all cases of diffuse tumour total resection of the superior maxilla was carried out and if necessary completed with resection of the malar bone, exenteration of the orbit and ethmoidectomy

In cases in which the tumour involved the posterior wall of the maxillary sinus and there was thus the possibility of invasion of the pterygopalatine fossa, surgery was always preceded by electrocoagulation of the tumour

19) All the patients underwent intracavitary radium therapy in doses varying according to the size of field to be irradiated Radium therapy generally covered a 3 7 day course with approximately 1 mcd per cm² of area to be irradiated with an effective dose

20) Within 20 30 days of completion of intracavitary radium therapy all the patients underwent external roentgenotherapy at the Turin University Radiological Department As high-energy sources were not available at the time, traditional roentgenotherapy was used. A daily tumour dose of 100 - 150 r was administered, the cumulative tumour dose amounting to not less than 3000 r

Within 2 3 months of completion of this first course of roentgenotherapy all the patients underwent a second course, generally providing a cumulative dose of 2000 r

Apart from radiation treatment of the primary lesion, in the 6 cases in which the laterocervical metastatic lymph nodes were removed roentgenotherapy of the cervical region was also carried out

21) The series reported consists of patients admitted between 1st May 1951 and 30th April 1957 It was thus possible to assess the results five years from commencement of treatment At the end of this five year period 18 of the 34 patients were alive and well and the other 16 had died

Three of these 16 patients died from causes unrelated to the tumour The other 13 patients died from causes closely related to the maxillary tumour Of these 1 had hepatic metastases 1 metastases to the iliac bone, 1 rib metastases and 1 laterocervical lymph node metastases and cancer of the neck

Of the 18 patients who survived the five year period and remained well without local neoplastic recurrence 5 died from causes unrelated to the maxillary tumour after respectively 6 years (2 cases), 7 years (2 cases) and 10 years (1 case).

Two patients died from causes closely related to the maxillary tumour 1 at 5 years 10 months (case 24) from hepatic metastases with no signs of local neoplastic recurrence, the other at 6 years 2 months (case 14) from pulmonary metastases following mastoid metastasis

The other 11 patients are alive and well after, respectively, 5 years (5 cases), 6 years (1 case), 7 years (1 case), 8 years (2 cases), 9 years (1 case) and 11 years (1 case)

22) With regard to five year survival in relation to histological type of tumour, of the 18 patients who survived the five year period 13 had differentiated carcinoma and 5 undifferentiated carcinoma

23) There was no evidence of a greater five year survival rate in patients in whom treatment was begun within 6 months of the onset of symptoms than in patients in whom treatment was begun at a later stage

24) The 18 patients who survived over five years from commencement of treatment include 7 cases of circumscribed tumour (out of 14 cases) and 11 cases of diffuse tumour (out of 20 cases)

The 4 cases of intrinsic tumour survived the five year period, as did 5 of the 12 cases of circumscribed extrinsic tumour The two patients of this group in whom exenteration of the orbit was performed died shortly afterwards from cachexia and local neoplastic recurrence

Only 3 of the 10 cases of diffuse extrinsic superior tumour survived the five year period, as did 6 of the 8 patients with diffuse extrinsic inferior tumour On the whole, better long term results were obtained in patients with diffuse extrinsic superior tumour

25) Invasion of the pterygopalatine fossa was observed in 13 of the 34 cases (2 cases of circumscribed extrinsic posterior tumour, 7 cases of diffuse extrinsic superior tumour and 4 cases of diffuse extrinsic inferior tumour) Only 4 of these 13 patients survived the five-year period Of these 4 patients 3 had diffuse extrinsic inferior tumours and 1 diffuse extrinsic superior tumour Of the 21 cases in which the pterygopalatine fossa was not invaded 14 survived the five year period

Our findings provide further evidence of the fact that invasion of the pterygo palatine fossa has a considerably unfavourable bearing on prognosis in malignant tumours of the maxillary sinus

26) None of the 6 patients with laterocervical lymph node metastases on commencement of treatment survived over five years

It may thus be concluded that the presence of laterocervical lymph node metastases verified clinically or histologically entails an unfavourable prognosis The finding of these metastases indicates that the tumour has spread beyond the anatomical limits of its site of origin and has entered a stage of widespread diffusion

REFERENCES

ANDERSON C A Malignancy of the antrum and ethmoid. *Med Times 71*, 180, 1942

ARONS I - Neoplasms of the antrum, nasopharynx and hard palate *Laryngoscope 51*, 61, 1941

ASHERSON N - Some experiences with radium in the treatment of malignant disease of the nose and upper jaw *J Laryng 44*, 739, 1929

ATKINS W L - Radiation and surgical treatment of malignancy in paranasal sinuses *New Orleans M & S J 94*, 19, 1941

BACKHOUSE T W WINDEYER B W The treatment of carcinoma of the maxillary antrum and ethmoids by supervoltage irradiation. *Proc Roy Soc Med 50*, 531, 1957

BACLESSE F Les cancers du sinus maxillaire, de l'ethmoïde et des fosses nasales *Ann Oto-laryng 69*, 465, 1952

BACLESSE F - ENNUYER A - CALLE R. Les epithéliomas du sinus maxillaire traites par roentgentherapie transcutanee seule *J Radiol 41*, 368, 1960

BALZARINI E. Il contributo della radiologia nel trattamento dei tumori del seno mascellare *Tumori*, 42, 152, 1956

BARNES H A The combined operative and radium treatment of malignant disease of the nasal accessory sinuses *Boston M & S J 183*, 648, 1920

BARROILHET J Antroscopy *J Laryng 59*, 231, 1944

BEATTY H S Cancer of the larynx and nasal accessory sinuses *Ohio State M J 43*, 843, 1947

BECK J C GUTTMAN M R Malignant diseases of nasal sinuses *Ann Otol 41*, 1, 1932

BECKER W MATZKER J Zur Diagnose und neuzeitlichen Therapie des Nasehnebenhohlenkarcinoms *Deuts med Wschnschr 80*, 1905 1955

BERGER E L BERGER M D Neoplasms of antrum *Laryngoscope 46*, 137, 1936

BISTOLFI F FILIPPI P BISTOLFI P MELAMPI M La stratigrafia assiale del cranio *Ed Cortina* Pavia 1959

BLUMLEIN H Die Rontgendiagnostik im Bereich von Nase und Nebenhohlen *Ztschr Laryngol Rhin Otol 33*, 149, 1954

BORDLEY J E LONGMIRE W P Rhinotomy for exploration of the nasal passages and the accessory nasal sinuses *Ann Otol 58*, 1055 1949

BOYSEN F Maligne svulster i nesen og dens bihuler *Nord med 28*, 2685, 1945

BOZZI E Carcinoma indifferenziato telangectasico del seno mascellare (diagnosi differenziale fra carcinoma simplex ed endotelioma) *Arch ital Otol* 59 222, 1948

BOZZI E MUCCHI L La tomografia nelle malattie dei seni paranasali *Ed Cappelli* Bologna 1947

BRAUND R R MARTIN H E Distant metastasis in cancer of the upper respiratory and alimentary tracts *Surg Gin & Obst 73*, 53, 1941

BRUN H Die malignen Tumoren der Nasennebenhohlen des Oberkiefers und des Unterkiefers *Prac oto-rhino-laryng* 18 171 1956

BRUNNER H Involvement of the paranasal sinuses in tumors originating from the mucous membrane of the cheek *Oral Surg Oral Med & Oral Path* 2 1235, 1949

CAPPS F C W Malignant diseases of the paranasal sinuses *Ann Roy Coll Surg England* 4 36, 1949

CAPPS F W WILLIAMS I G Discussion on malignant diseases of the nasal cavity and sinuses *Proc Roy Soc Med* 43 665 1950

CARREGA N LEGRES CLEMENT R A propos de 54 cas de tumeurs malignes du sinus maxillaire traites au C A C de Marseille de 1934 à 1956 *J Radiol* 41 364 1960

CAVALLOT A MONATERI P C La diagnostica differenziale radiologica delle alterazioni dei seni mascellari *Min O rinolaring* 4 168 1954

CERONI T BARAVELLI P Problemi diagnostici e terapeutici in tema di neoplasie maligne del massiccio facciale *Arch ital Otol* 71, 289, 1960

COLLINS V P POOLE J L Treatment of antral cancer by combined surgery and radium therapy *Radiology* 55 41 1950

Two patients died from causes closely related to the maxillary tumour 1 at 5 years 10 months (case 24) from hepatic metastases with no signs of local neoplastic recurrence, the other at 6 years 2 months (case 14) from pulmonary metastases following mastoid metastasis

The other 11 patients are alive and well after, respectively, 5 years (5 cases), 6 years (1 case), 7 years (1 case), 8 years (2 cases), 9 years (1 case) and 11 years (1 case)

22) *With regard to five year survival in relation to histological type of* tumour, of the 18 patients who survived the five year period 13 had differentiated carcinoma and 5 undifferentiated carcinoma

23) There was no evidence of a greater five year survival rate in patients in whom treatment was begun within 6 months of the onset of symptoms than in patients in whom treatment was begun at a later stage

24) The 18 patients who survived over five years from commencement of treatment include 7 cases of circumscribed tumour (out of 14 cases) and 11 cases of diffuse tumour (out of 20 cases)

The 4 cases of intrinsic tumour survived the five year period as did 5 of the 12 cases of circumscribed extrinsic tumour The two patients of this group in whom exenteration of the orbit was *performed died shortly afterwards from* cachexia and local neoplastic recurrence

Only 3 of the 10 cases of diffuse extrinsic superior tumour survived the five year period, as did 6 of the 8 patients with diffuse extrinsic *inferior tumour* On the whole, better long term results were obtained in patients with diffuse extrinsic superior tumour

25) Invasion of the pterygopalatine fossa was observed in 13 of the 34 cases (2 cases of circumscribed extrinsic posterior tumour, 7 cases of diffuse extrinsic superior tumour and 4 cases of diffuse extrinsic inferior tumour) Only 4 of these 13 patients survived the five-year period Of these 4 patients 3 had diffuse extrinsic inferior tumours and 1 diffuse extrinsic superior tumour Of the 21 cases *in which the pterygopalatine fossa was not invaded, 14 survived the five year* period

Our findings provide further evidence of the fact that invasion of the pterygo palatine fossa has a considerably unfavourable bearing on prognosis in malignant tumours of the maxillary sinus

26) None of the 6 patients with laterocervical lymph node metastases on commencement of treatment survived over five years

It m [illegible] us be concluded that the presence of laterocervical lymph node metastase [illegible] d clinically or histologically, entails an unfavourable prognosis The finding [illegible] astases indicates that the tumour has spread beyond the anatomical lim [illegible] site of origin and has entered a stage of widespread diffusion

GROSSMAN A A DONNELLY W A SNITMAN M F - The management of malignancy of the maxillary sinus *Canad M A J* 62, 576, 1950

HAJECK M Die Erfolge der kombinierten operativen Behandlung und Strahlentherapie mittels Radium an der laryngo-rhino-otologischen Klinik wahrend der lezten 10 Jahre *Wien klin Wschr* 42, 1653, 1929

ARBERT F Unusual neoplasm of the antrum. *U S Navy M Bull* 41, 1405, 1943

ARMER W D - Treatment of malignant disease in the upper jaw *Lancet* 1, 129, 1935

EATLY C A - Expanding tumors of the maxillary sinus value of lateral rhinotomy *New York J Med* 51, 2640, 1951

EATLY C A - Tumors of the maxillary sinus *Oral Surg* 5, 1253, 1952

EATLY C A - Clinical aspects of tumors of maxillary sinus *Arch Otolaryng* 57, 152, 1953

EATLY C A Tumors of the maxillary sinus *Arch Otolaryng* 57, 153, 1953

EMENWAY W G - LINDSAY J R Malignancies of paranasal sinuses *Arch Otolaryng* 70, 61, 1959

ENDRICK J W Treatment of cancer of the nasal cavity and paranasal sinuses *Surg Gyn & Obst* 102, 322, 1956

ENDRICK J W - Treatment of cancer of the paranasal sinuses and nasal fossa *Arch Otolaryng* 68, 604, 1958

HENSCHEL CH Tumeurs malignes du nez et des cavites paranasales *Acta O R L Belgica* 5, 428, 1951

HOLMGREN G - Erfahrungen uber chirurgische Behandlung von malignen Oberkiefertumoren *Acta oto-laryng* 7, 511, 1925

HOLVEY E H ROSENTHAL L M Tomography in the region of the maxillary sinuses *Radiology* 42, 458, 1944

HOMMERICK K W Metastasenhaufigkeit bei bosartigen Oberkiefergeschwulsten *Arch Ohren Nasen u Kehlkopb* 169, 459, 1956

HOUSER K M Diagnosis and treatment of primary malignant neoplasms of the maxillary sinus *Arch Otolaryng* 18, 643, 1933

HUET P C STEFANI S Les cancers du massif maxillaire superieur Etude anatomo-clinique et therapeutique *Ed Masson*, Paris, 1960

HUGHES H A ARCHER R H Internal irradiation of antral and mastoid cavities *Brit J Radiol* 25 152, 1952

HUIZINGA E Diagnostiche Schwierigkeiten bei malignen Nasennebenhohlentumoren *Acta oto-laryng* 25 296, 1937

UHLMANN F RATING B SCHNEIDER W Die Bedeutung der Schnittaufnahme fur die Diagnostik der Nasen Nebenhohlenerkrankungen *Arch f Ohren, Nasen, u Kehlkopfb* 148 252, 1940

USTNER W Zur Rontgendiagnostik der Kieferhohlenerkrankungen unter Verwendung von Kontrastmitteln *Deutsche Stomatol* 4, 209, 1954

BAYLE J ROTH F L'anesthesie generale et la chirurgie exsangue dans les interventions pour tumeurs des maxillaires *Sem Hop* 28, 1304, 1952

SSON L G MARTENSSON G Carcinoma der Nasennebenhohle und Nasenschleimhaut. *Acta Oto-laryngol suppl* 116, 172, 1954

SSON L G MARTENSSON G Carcinoma of the paranasal sinuses and the nasal cavities A clinical study of 379 cases treated at Radiumhemmet and the otolaryngologic department f Karolinska Sjukhuset 1940-1950 *Acta Radiol* 42, 149 1954

ER F L Cancer of the nasal accessory sinuses problems on diagnosis and management *rb Phys Therapy* 16, 199 1935

Malignant neoplasms of the nose, nasopharynx and paranasal sinuses evaluation of n therapy *Tr Am Acad Ophth* 55, 214 1951

G B MEDA P I tumori maligni delle prime vie aeree e digestive nell'eta *Arch Otol Rinol Laringol* 64, 540 1953

ERT J ENNUYER A CALLE R Cancers du massif ethmoido-maxillaire. Classification o-clinique Modes de traitement chirurgicaux et radiochirurgicaux Resultats (Statisti 104 cas) *Ann Oto-laryng* 68 617, 1951

The calculation of the dosage in the radium treatment of carcinoma of the antrum *et & Mississippi Valley M J* 59 56, 1937

NCI N I tumori maligni del massiccio facciale *Casa Ed Ambrosiana* Milano

MAJER E H Zur Behandlung der Nasenn b le (rz ro n ur.t 4 216 1949

MARQUES P BIRAGUF C HEAIOUS G M ta t. ut nécs pithelioma du maxillaire superieur *Bull Ass fr et cancer 47* 389 195

MARQUEZ P BRU A DELPA A. Irra lit on ar f rap des rs du sinus maxillaire *J Radiol 39* 134 1958

✓ MARTIN H E The fractional or divided dose d of external ti ion in the treatment of cancer of the pharynx tonsil larynx and s sl sinuses *A of 16* 1 1935

✓ MARTIN H Cancer of the head and neck I 1306 1948

MATTICK W L STREUTER M A Carcinoma of t antrum *Surger 35* 236 1954

MEDA P Le syndrome neuro-l dol gique du cancer ethmoido-maxillaire *J fr O R L 2* 282 1953

MOFFET A J The origin nd treatment t cases of the upper jaw and nasal sinuses *J Laryng 55* 132 1 52

MORRISON L F Rad. cobalt in o ngolo 52 153 1951

MOTTA G BARAVELLI P CER T Osseo et s malignant tumours of the maxillo-ethmoidal region ta O pl 1 1 61

MYERSON M C Tl m ncer the na l sinuses *Laryngoscope 48* 615 1938

NEW G B Mal g n the n ubgmere *Arch Otolar 4* 201 1926

NEW G B Tre r of the upper jaw and antrum fifteen years of progress *Su g* 6 1

NEW G B Car ab lity malignan tumors of the upper jaw and antrum *Tr 1 n Li 41* 5 1935

NEW G B bilities of malignant tumors of the upper jaw and antrum *Surg G*

NEW G B of the paranasal sinuse *m J Surg 42*, 170 1938

NIEDE or Carcinom nd Trauma *lr d Wschr 10* 688 1955

OH f the following electrocoagulation treatment of cancer of the *16* 2 1931

C umors of the maxillo-ethmoidal region clinical study with special lectros gery and irradiation *Acta oto-laryng suppl 19* 1 1933

OHNGREN G Malignant disease of the upper aw *J Laryng 52* 8 1937

O I J J CLERI L M Malignant umors of the maxillary sinus analysis of 47 fatalities *Ann Otol 55* 312 1946

O HEIM O Cancer of nose nd nasal sinuses *Tidsskr norske laegefor 70* 448 1950

PALETTA I WEIR D C Malignant tumors of the paranasal sinuses *Surgery 43* 752 195

PETZIK S L Management of cancer of the nasal cavity and paranasal sinuses *California Me 74* 374 1951

PETTERINO-PATRIARCA A Il trattamento elettrochirurgico dei tumori maligni del massiccio f ciale *Oto-rino-laring ital 8* 429 1938

PFAHLER G E VASTINE J H jr Roentgen diagnosis of cancer of the accessory sinuses *Ar Otolaryng 31* 561 1940

PFANDER F Beobachtungen und Behandlungensergebnisse bei 102 maligne Tumoren der N und Nebenhohlen *Arch Ohren Nasen u Kehlkopfh 168*, 133 1955

PIETRANTONI L « Trattamento chirurgico dei tumori maligni del massiccio facciale » *Calicetti P Trattato di chirurgia otorinolaringologica* Ed Cappelli Bologna 1940

PIETRANTONI L Trattamento chirurgico ed elettrochirurgico dei tumori maligni delle vie digestive *Relazione V Congresso Lega Italiana per la lotta contro i tumori* Ed Mar Pisa 1947

PIETRANTONI L Zone nevralgiche e zone di anestesia della regione facciale e della cavit come sintomi precoci di alcune forme di tumori maligni delle cavità paranasali *Ar Otol 59* 105 1948

PIETRANTONI L FREGNI R Epitheliomas ethmoido-maxillaires et epitheliomas de l an taire interne a evolution orbitaire *Ann Oto-laryng 76* 601 1959

PIETRANTONI L Le cancer du maxillaire superieur Aspects cliniques et possibilites tiques Resultats apres 5 ans sur 113 cas operes *Fortschr der H N O VI* Basel

POLLACK R S Carcinoma of the maxillary sinus *Ann Sur* 5 7

PONTHUS P DARGENT M PAPILLON J Sur les possibilit therapie dans du sinus maxillaire *J Radiol 26* 73 44-45

PORTMANN G - Considerations sur le traitement chirurgical des tumeurs malignes du massif facial *C R Soc fr O R L 56*, 56, 1949

PORTMANN M GUILLEN G Interet de la tomographie horizontale dans le diagnostic des tumeurs des sinus de la face *Rev Laryng 75*, 59, 1954

POULSEN Y B Malignant tumors in nose and paranasal sinuses *Acta oto-laryng 43*, 474, 1953

QUICK D Treatment of malignant growths of the nasal accessory sinuses and nasopharynx *Radiology 14*, 191, 1930

RAINES D JAMES A G The menagement of cancer of the maxillary antrum *Surg Gyn & Obst 101*, 595, 1955

RATKO A L Tumors of the nose and paranasal sinuses *J Intern Coll Surgeons 25* 777 1956

RATTI A Contributo radiologico allo studio dei tumori maligni del seno mascellare *Radiol med 22* 1, 1935

RINGERTZ N Pathology of malignant tumors arising in the nasal and paranasal cavities and maxilla *Acta oto-laryng suppl 27*, 1, 1938

ROBIN I G Malignant disease of the maxilla with special reference to carcinoma of the antrum A review of the literature, with an account of the cases admitted to Guy s Hospital over a ten year period *Guy s Hosp Rep 89*, 301, 1939

ROBINSON G A A Diagnosis and treatment of malignant tumors of the nasal sinuses *Am J Roentgenol 34* 641, 1935

ROBINSON G A Diagnosis, treatment and end results in malignant tumors of the nasal sinuses *New York J Med 44* 713 1944

ROSSI G MENZIO P Contributo alla conoscenza dei reticolo-sarcomi primitivi del naso *Il Valsalva 30* 212, 1954

ROSSI G Studio anatomo-clinico su 56 casi di tumori maligni della regione faringo tonsillare *Giorn Acc Med Torino 117*, 218 1954

ROSSI G I tumori maligni della regione faringo-tonsillare *Arch ital Otol 67* suppl 28 1956

ROSSI G CHERUBINI E Malignant tumors of the pharyngo-tonsillar region A study about forty two cases *Acta oto-laryng 52* 477 1960

SCHALL L A Malignant tumors of the nose and nasal accessory sinuses *J A M A 137* 1273 1948

SCHALL L A Malignant neoplasms of the nose paranasal sinuses and nasopharynx evaluation of surgical treatment *Tr Am Acad Ophth 55* 209 1951

SCHUKNECHT H F Surgical menagement of carcinoma of the paranasal sinuses *Laryngoscope 61* 874 1951

SCHWAB W WERNER K KAESS H Erfahrungen dei der Behandlung maligner Nebenhohlen geschwulste *Strahlentherapie 101* 227 1956

SEED G S Malignant tumors of the upper jaw *J Fac Radiologists 2* 263 1951

SEELIG C A Carcinoma of the antrum *Ann Otol 58* 168 1949

SEGUINARD M Les metastases des epithéliomas du massif facial supérieur *Rev Laryng 73* 481 1952

SVELLING M D *Discussion on the radiation treatment* of cancer of the antrum and eth[illegible] *Proc Roy Soc Med 50* 529 1957

STRUBEN W H Malignant tumours of paranasal sinuses Treatment and results *Ann Otol [illegible]* 754 1957

SZENDE B Die bosartigen Geschwulste der Nasennebenhohlen *Monatschr f Ohrenh [illegible]* 925 1938

SZPUNAR J Treatment of malignant tumors of nasal sinuses *Acta oto-laryng [illegible]*

TABB H G Carcinoma of antrum Analysis of 60 cases with special reference to [illegible] exirpation *Laryngoscope* 67 269 1957

TABB H G Symposium Modern criteria for diagnosis and therapy of malignant [illegible] upper respiratory tract the nasal cavity and paranasal sinuses *Laryngoscope [illegible]*

TABB H G Maxillectomy in carcinoma of the antrum An analisis of 80 cases [illegible] certain modifications of the basic technique of exirpation *Laryngoscope [illegible]*

TAKASHI M Carcinoma of paranasal sinuses its histogenesis and classification *[illegible]* 32 501 1956

THIESSING G Ein Beitrag zur Frage der Schadigungen durch Kontrastfullung [illegible] *Arch f Ohren Nasen u Kehlkopfh 151* 356 1942

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

ACTA OTO-LARYNGOLOGICA
SUPPLEMENTUM 182

P L E M E N T U M 182

ON THE FUNCTION OF MIDDLE EAR AND EUSTACHIAN TUBE

Eight reports

From the Department of Otolaryngology
(Head: Prof. Hj. Koch, M.D.), University of Lund, Sweden

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM O

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

ACTA OTO-LARYNGOLOGICA
SUPPLEMENTUM 182

ON THE FUNCTION OF MIDDLE EAR AND EUSTACHIAN TUBE

Eight reports

From the Department of Otolaryngology
(Head Prof Hj Koch, M D), University of Lund, Sweden

LUND, 1963

Malmö 1963
Sydsvenska Dagbladets Aktiebolag
[illegible]

CONTENTS

INTRODUCTION

When Professor Zollner and Professor Wullstein, the famous German otologists, published their first works on reparative middle ear surgery in the early 1950's, it was obvious that a new era in otology had been ushered in Otologists from all over the world went to Freiburg and Ziegen in order to study the methods developed and the instruments used there. From the very beginning the German researchers had stressed the fact that a normal tubal function and an intact cochlea are prerequisites for a successful result of the operations. Nevertheless, diagnostics have often been unsatisfactory particularly with regard to the tubal function. This may in part explain the numerous failures to obtain improvement of hearing in cases of chronic otitis Such failures have been reported from all parts of the world in later years. In addition, the methods so far available for testing the tubal function only admit of a qualitative determination of the middle ear ventilation through the tube. These methods have largely been worked out by Zollner himself Reliable quantitative methods, however, have hitherto been lacking although such methods must be considered highly important for judging the degree of operability At the ENT Clinic of Lund methods have been worked out which allow both a qualitative and a quantitative determination of the tubal function Furthermore, methods have been evolved for the determination of the volume of the air filled ear system and of the degree of mobility of the drum.

The present supplement is a comprehensive account of the research work of the clinic in these fields. A number of problems still remain unsolved. It is hoped that this volume will be a stimulus to further research on the middle ear and the Eustachian tube.

Lund, March, 1963
Hjalmar Koch

QUALITATIVE TESTING OF THE EUSTACHIAN TUBE FUNCTION

SVEN INGELSTEDT and URBAN ÖRTEGREN

Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch, M.D),
University of Lund

New methods for qualitative testing of the Eustachian tube function have been devised. A threshold pressure device has been constructed by which required threshold negative or overpressures can be produced within the rhinopharynx during deglutition without giving any troublesome blowing of air through the nose. The pressure range produced by the device ranges from —40 to +40 mm Hg The tubal air passage is indicated with the aid of a flow rate meter device opening into ambient pressure. This is connected to a cuff system which is airtightly inflated with glycerine when lodged selectively within the bony part of the ear canal. During the tubal air passages small air puffs are developed in the external ear canal, which are recorded as an air flow Thanks to the high sensitivity of the flow meter device the method can be used for cases with both intact and perforated ear drums. These methods are intended for clinical tubal function studies, and various tubal function tests have been carried out.

During the last ten years, studies on the tubal function have become increasingly important from a clinical point of view as a pre-examination in connection with surgical restoration of the sound conduction system in chronic otitis A pre-operative testing of the tubal function seems to be quite as important as an audiogram for the choice of cases suitable for operation and for selecting the optimal operation technique It seems highly remarkable that no information should have been given about the pre-operative state of the tubal function when the results of middle ear surgery have been reported.

Many different studies on methods for such testing have been published. From a methodological point of view they have been divided into two main groups, i.e qualitative and quantitative methods The qualitative ones only afford information as to whether the tube is open or closed. On the other hand, the methods are called quantitative either when they provide some estimation indirectly by determining the intra-tympanic pressure or when the resistance of the tube is directly graded from known threshold pressures produced within the rhinopharynx For literature in this field the reader is referred to the extensive references of Zollner (1942) and Thomsen (1955, 1957) To judge from earlier investigations, however, hardly any of the methods used seems sensitive enough to allow an application both on ears with intact and with perforated drums, which is necessary for the method to be really useful clinically. Therefore, the present authors worked out more accurate methods suitable for clinical investigations on the tubal function

Earlier methods

For practical reasons, the methods can be divided into two different groups methods giving tubal passage and methods indicating tubal opening

Based on a paper read before the Swedish Otorhinolaryngological Society on May 19, 1961

As regards the indication methods for tubal air passage, inspection is generally used clinically for intact drums and auscultation in the ear canal for perforated ones These methods have doubtless a clinical value in spite of being subjective Objective recording of the tubal function, however, is a prerequisite for further progress in this field In order to be useful clinically the indication methods must be equally applicable in cases of normal and perforated ear drums Such a method has been worked out by Oltersdorf (1962) But all manometer systems used for direct indication have hitherto been connected only to the membranous part of the external ear canal The least movement of the canal causes heavy disturbing pressure fluctuations during the recording, which are magnified by the use of closed-system manometers Again, there is the risk that disturbing pressure fluctuations may arise owing to thermal influence on the enclosed manometer gas In spite of this, Oltersdorf is of the opinion that it is possible to calculate the tubal ventilation even quantitatively by measuring the angle between the pressure curve deflections and the base line on the ear manometer recordings Kottmeyer has described a plethysmograph method, implying that the whole of the ear canal is filled with fluid This, however, has a mass increasing effect on the drum

Test of the Valsalva and Politzer types, sound conduction through the tube, catheters, and X-ray studies only provide information as to whether the tube is open or not, but do not tell us whether the tube can meet normal physiological demands This last mentioned point is an important one for the clinical usefulness of tubal function tests

A positive Toynbee manoeuvre is generally considered valuable for an estimation of the normal tubal function because deglutatory movements are needed during the test This test is unfortunately negative, i e it gives no tubal air passage in about 30 % of normal cases according to Zollner and Thomsen

In addition to pressure drop determinations across the tube, real quantitative measurements of the tube's resistance to air flow should provide simultaneous flow rate determinations The determinations of the resistance of the tube performed so far have not included measurements of the flow rate Consequently, the present paper has intentionally been entitled "qualitative testing"

According to the present authors the following demands must be satisfied by a clinical method constant square pressure waves must be produced within the rhinopharynx during tubal testing moments and must not cause the patient any discomfort by gas flow The desired threshold pressure level must be produced both above and below the ambient pressure and should be adjusted in advance Every uncontrolled pressure change within the closed rhinopharynx during testing moments should be recorded Every tubal air passage should be indicated and recorded simultaneously without causing the patients any discomfort The indication method must be quite as sensitive when indicating movements of the normal ear drum during tubal air passage as when applied to perforated drum cases Disturbing influences on the curves recorded, indu-

ced by movements of the ear canal, should be reduced to a minimum. The physical data necessary for estimating the accuracy of the method used must first be obtained in model experiments and must be published

Present Investigation

Methods

Threshold pressure device

The pressure changes within the closed rhinopharynx needed for tubal air passage are produced with the aid of an ordinary vacuum cleaner functioning either as a sucking or a blowing fan The blowing or sucking capacity of the fan is regulated with a variable autotransformer (see Fig 1). The system includes an air container, volume 10 liters, functioning as a pressure equalizing air chamber From the container runs a plastic tube (inner diameter 12 mm) to a nose piece which can be airtightly connected to one of the subject's nostrils In this tubing system an electromagnetic valve is mounted between the container and the nose piece, and this valve can be opened when manually

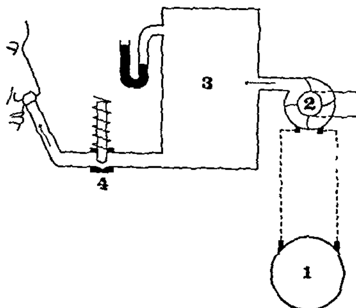


Fig 1 Threshold pressure device connected to the nose 1 Autotransformer 2 Vacuum cleaner 3 Air container 4 Electromagnetic valve

governed by the examiner. By means of a water manometer connected to the air container the desired pressure can be adjusted in advance by the fan blowing into or sucking from the closed 10-liter system The pressure obtained ranges from 0 to ±40 mm Hg

Pressure control system for the rhinopharynx

A pressure transducer of a variable inductance type is airtightly connected to the free nostril by a nylon tube, inner diameter 1 mm, and a nose piece The transducer is of a standard type, pressure range 0 to 300 mm Hg (EMT 490 A, Elema Schonander AB, Stockholm) This transducer is regularly calibrated for known pressures with an ordinary water manometer For further calibrations during the course of each investigation a standardization push button is available In this pressure transducer the pressure is transformed into voltage which is conducted into an amplifier unit A direct writing ECG apparatus (Mingograph, Elema Schonander AB, Stockholm) is connected to the output of the amplifier The sensitivity of the manometer system is so adjusted that a pressure change of 10 mm Hg gives a recorded deflection of 30 mm. The frequency response curve for the electromanometer appears in Fig. 4

Model experiments

First an experimental set up is made for the determination of the pressure equilibration time after the application of a threshold pressure to the closed nose and rhinopharynx during a deglutition A pressure of 5 cm water level is built up in the closed 10 liter system and the nose tube is airtightly connected to another closed container, volume 500 ml, at atmospheric pressure After momentary opening of the valve a determination can be made of the time needed for pressure equilibration in the greater volume to the same level as that previously adjusted in the original volume A steady pressure level of 5 cm water is reached in the whole system within 1/10th of a second It should then be observed that the size of the connected volume may be estimated at about five times the total closed volume of nose and rhinopharynx during swallowing

Secondly, before starting any pressure recording experiment it is necessary to make sure that the response of the instrument is rapid enough for recording the phenomenon in question Therefore, by simultaneously comparing the effect of variation in the dynamic responses of two different manometer systems (10 and 20 cps) on the pressure changes produced in the rhinopharynx during the same Toynbee test, it is shown that a manometer system with a uniform response up to 10 cps provides adequate reproduction of all intra aural and rhinopharyngeal pressure pulses

The performance of threshold pressure experiments

The pressure generator device is airtightly connected to one of the nostrils and the electromanometer for controlling threshold pressure in the closed rhinopharynx is connected to the other one The patient respires through the mouth The pressure is adjusted to a desired level and while he swallows some water the electromagnetic valve is momentarily opened At the same time the pressure is equilibrated to that in the container within the closed space of the

nose and the rhinopharynx. A steady pressure level is built up within the rhinopharynx as seen from the manometer recordings during testing. There is no air flow through the nose between the testing moments.

Indication device for tubal air passage

On principle this system consists of an air flow rate meter airtightly connected by a small nylon tube (inner diameter 1 mm) to the inner part of the bony external ear canal. The bony canal is hermetically sealed by a rubber

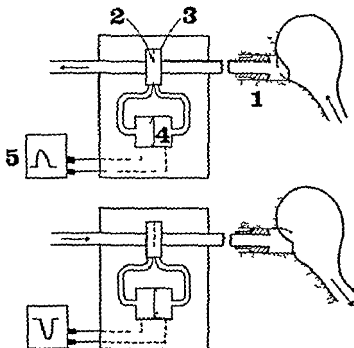


Fig. 2. Flow rate meter device for tubal air passage indication. 1. Cuff system. 2. Flow resistor. 3. Cylinder with pressure taps. 4. Differential pressure transducer. 5. "Outward flow" recording.

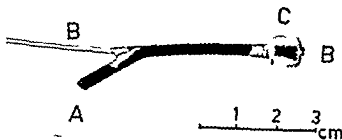


Fig. 3. Rubber cuff system. A. Glycerine inflation tubing. B. Pressure recording tubing. C. Cuff.

cuff system (see Fig 2) The air puffs, caused by the movements of the intact drum or by an air leakage through a perforation during a tubal air passage, are measured as an air flow

The cuff system consists of a small rubber cuff with a nylon tube (inner diameter 1 mm) opening into the closed system behind the inflated cuff The cuffs are manufactured by the authors and are inflated with glycerine (see Fig 3) This inflation can be performed without causing any discomfort, and no local anesthesia is needed After filling the cuff placed in the bony canal, the tightness of the system is regularly checked with a water manometer and endures a maximum pressure of up to 40 mm Hg When the cuff system is applied correctly it has no contact whatever with the membranous ear canal, nor of course with the drum The subject is sitting in an examination chair with his head firmly fixed

The flow rate meter device

If an air resistance in a tube is placed in a flow stream, this resistance causes a difference in pressure between its upstream and downstream sides The actual resistor consists of a Polyvinyl chloride filtre with small uniform pores The filtre is mounted across a brass cylinder, inner diameter 10 mm (see Fig 2) On both sides of the resistor pressure taps are mounted which are then connected to a differential pressure transducer detecting the pressure difference across the resistor during the flow Working pressure range for the differential pressure transducer —50 to +50 mm water level (Pneumomanometer EMT 573, Elema Schonander AB, Stockholm) The pneumomanometer is of a standard type, and primarily constructed for pneumotachography recording The inner air volumes of the differential manometer + the flow resistor down to the membrane and the filtre are made exactly alike, i e 1 8 ml on both

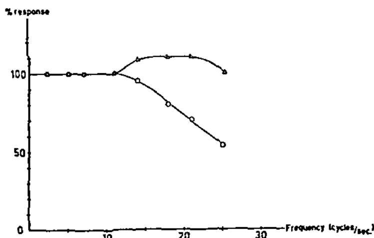


Fig 4 Frequency response curves for the manometer devices —△—△—△—=manometer for recording pressure variation in the rhinopharynx. —O—O—O—=flow rate meter device for tubal air passage indication

sides The sensitivity of the flow meter may be illustrated as follows if a momentary volume flow of 1 mm³ is produced, a deflection of 8 mm is recorded The frequency response curve for the flow rate meter system appears from Fig 4 The differential output of the manometer is amplified and fed into one channel of an ECG apparatus It appears from the recordings that the upper curve shows the indication of the tubal air passage (M) and the lower curve the pressure changes in the closed rhinopharynx simultaneously (R) With outward movements of the drum or outward blowing through the perforation the recording curves run upwards, while inward movements of the drum give curves running below zero. Pressure recordings for the rhinopharynx overpressure is recorded as upward curves and negative pressure as downward curves

Results

Fig 5. Three normal cases making different shapes of pressure changes within the rhinopharynx during Toynbee manoeuvres (R) and the resulting *pressure change movements of the ear drums (M) Two different pressure* shape curves are shown the biphasic and the purely negative types of pressure waves in the rhinopharynx The arrows in the (M) curves indicate the moment of "open nose" deglutition.

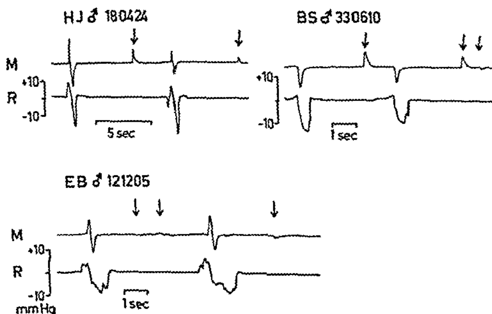


Fig 5 Positive Toynbee manoeuvres in three normal ear cases M=tubal air passage indication R=rhinopharyngeal pressure recordings. ↓ ="open nose" deglutitions.

Fig 6 Illustrates positive Toynbee manoeuvres in two cases of chronic otitis

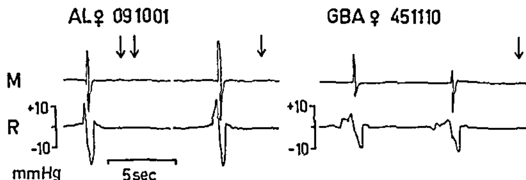


Fig 6 Positive Toynbee manoeuvres

Fig 7 Shows high speed recordings of Toynbee tests in two cases Observe that the tube is closed already long before the negative pressure in the rhinopharynx has reached its maximum

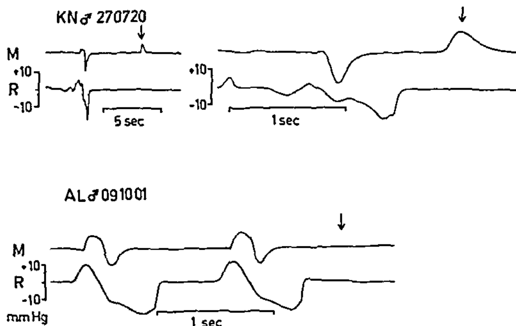


Fig 7 High speed recordings during the Toynbee manoeuvres. The curves in the upper half are obtained from a normal case the curves in the lower half from a case of chronic otitis.

Fig 8 Shows typical recordings of Valsalva and Politzer tests in a normal case and in a case of chronic otitis The arrows indicate "open nose" deglutitions by which the middle ear overpressure again falls to ambient pressure moving the distended ear drum back to its normal position

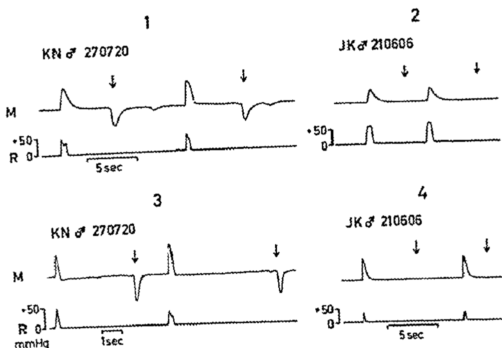


Fig 8 Positive Valsalva and Politzer test recordings. 1—2 Valsalva (1 normal, 2 chronic otitis) 3—4 Politzer (3 normal 4 chronic otitis)

Fig 9 Negative and positive square wave threshold pressure levels are produced in the rhinopharynx during deglutition The M curves indicate tubal air passage in both flow directions following the pressure recorded in the rhinopharynx (R)

Fig 10 Tuba aperta A 55 year old woman without any history of body weight loss. She was sent to hospital by her family, who was seriously disturbed by her continuous daily loud sniffing for about two years She felt fullness of the right ear disappearing for some moments after every sniff manoeuvre No autophony Otoscopy and microscopy revealed a normal, immobile drum. The figure illustrates how the drum moves (M) synchronously with the respiratory pressure changes within the rhinopharynx (R), recorded during

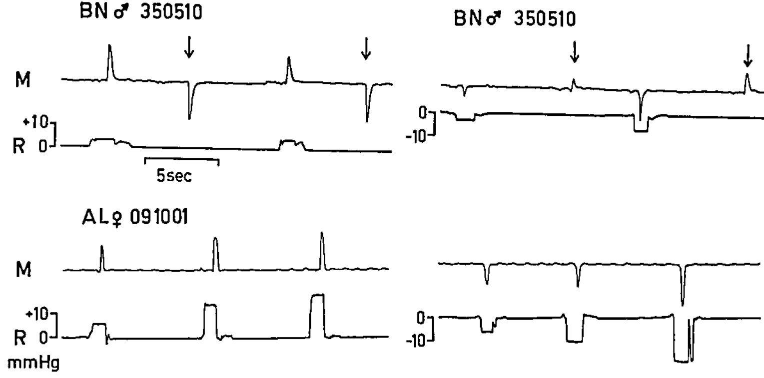


Fig 9 Threshold pressure application (R) giving tubal air passages in both flow directions (M). Normal ear case (upper half). Chronic otitis (lower half).

nasal respiration When the patient sniffs the tube is closed by sucking and the drum movements disappear After each swallowing the tube opens again (see the arrows)

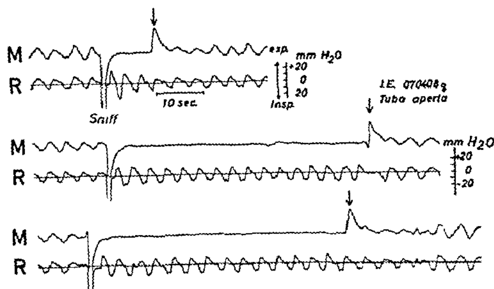


Fig 10 A patulous tube ear case M=ear canal indication recordings R=respiratory pressure changes recorded in the rhinopharynx during nasal respiration ↓ ="open nose" degluti ons.

General Discussion

The threshold pressure device sketched above is *100 %* safe because it contains no mechanical valve system, the fan itself functioning like a kind of valve A further advantage is that the mucous membranes in the nose and rhinopharynx are not irritated by any air blowing, which may interfere with the normal act of swallowing This device is not only suitable for diagnostic purposes but can also be used therapeutically for graded middle ear insufflations

In his monograph, Zollner points out that it is too complicated clinically to shut off the external ear canal for the purpose of tubal air passage indication However according to the present authors this is a comparatively simple procedure but the cuff system used must then be applied to the bony part of the ear canal only This is the only way of obtaining safe recordings of the tubal air passage thereby avoiding any disturbing pressure influences caused by the movements of the membranous part of the ear canal where all cuff systems hitherto described have been lodged Furthermore, several authors have used closed manometer systems for this indication, thereby making it even more difficult to obtain accurate recordings since the thermal influence on the enclosed manometer gas gives pressure changes Finally, a

counterpressure may be built up in the closed manometer system, which interferes with the drum movements to be studied Thus the advantages of using a system of the flow rate meter type, which opens into ambient pressure, are obvious

Most authors think that auscultation in the auditory canal or inspection of the drum movements with a microscope are test procedures sensitive enough for clinical indication of tubal air passages According to the present authors, however, these methods are very often too insensitive even for clinical use The correctness of this statement appears from the following discussion

If an air passage can be demonstrated during the Toynbee manoeuvre the function of the tube is generally considered normal This test, requiring no special apparatus, is widely used An analysis of its value for clinical examinations is therefore of interest

Already in 1865 Politzer pointed out that rapid pressure changes with both positive and negative phases develop during swallowing when the nose is closed, with an amount of pressure in the initial positive phase of +5 to +10 mm water column, followed by a negative one of —60 to —120 mm Perlman (1951) reports the corresponding amount of pressure from +100 to —100 mm water column In the present investigation, comprising more than 100 patients, the actual pressure variations obtained in the rhinopharynx ranged from +140 to —280 mm water column This amount of pressure is greater than that found in earlier investigations, probably thanks to the more accurate pressure transducer technique used Now, most authors report that the physiological range of pressure difference between the middle ear and the rhinopharynx needed for air passage through the normal tube during deglutition is from about +70 to —70 mm water column Consequently, as regards the amount of pressure recorded in the rhinopharynx, the tube only should be tested, at least during the negative phase of Toynbee, at a pressure range which is nearly always far below the given lower physiological pressure limit

The shape of the pressure variations recorded during Toynbee may vary in degree and type not only from subject to subject but also in each subject depending on dry or water swallowing

Next, according to the literature a Toynbee experiment results in different types of air passage through the Eustachian tube Toynbee (1853) considered that air was pressed into the middle ear during the manoeuvre Politzer reported that initially during the manoeuvre a slight overpressure was produced in the middle ear, which was replaced by a more pronounced negative pressure When recording the drum movements with an ear canal manometer, Perlman (1951) found two significant types of drum movement either a sucking in of the drum only, or a sucking in immediately followed by an outward movement during which the drum returned to its normal position Perlman concluded that the tube does not open during the initial period of positive pressure recorded in the rhinopharynx Finally the Toynbee manoeuvre fails to give any tubal air passage in about 30 per cent of normal cases according to Zollner

and Thomsen Consequently, further studies in this field are required it is necessary to record all pressure changes developed in the rhinopharynx simultaneously with objective indication of the resulting tubal air passages The amount and duration of the pressure variations, recorded in the rhinopharynx, must be determined in relation to the period during which the tube is kept open

From the present investigation it appears that the Eustachian tube is only kept open during part of the time needed for the recording of all the pressure changes produced in the rhinopharynx during swallowing with the nose closed The type and degree of middle ear ventilation, which may then follow, is entirely dependent on the amount and the type of the pressure changes actually taking place in the rhinopharynx during the tubal opening period These result in varying types of recorded ear indication curves, although the simultaneously recorded pressure complexes in the rhinopharynx may all have similar curve shapes during the experiments.

Fig 11 Shows the most commonly recorded types of rhinopharyngeal pressure complexes to the left and the resulting tubal air passages to the right Six

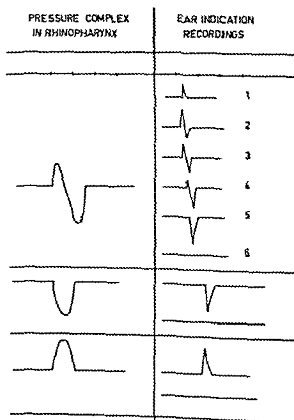


Fig 11 Various types of pressure change and air passage indication recordings obtained during Toynbee manoeuvres.

different types of tubal ventilation recordings can then follow a biphasic pressure complex. The commonest type is number 4 followed by 5, both resulting in a final negative middle ear pressure. Unfortunately, however, the Toynbee manoeuvre is reported not to give any tubal air passage at all in several normal ear cases. Such a negative result as number 6 is very seldom recorded in the present normal ear cases at repeated manoeuvres, and is probably at least in part dependent on the techniques used for the indication of tubal air passage. In many of the normal cases the present authors could not indicate any tubal air passage by microscope inspection of the drum although a tubal air passage could be nicely recorded. In several cases, furthermore, the middle ear cavity is insufflated and exsufflated with the same volume of air during one single Toynbee manoeuvre (number 3). Thus the ear drum has first made an outward movement from the normal position, immediately followed by an inward movement during which the drum returns to its normal position. This phenomenon may possibly in part explain why 28 per cent of the Toynbee manoeuvres failed in Thomsen's material of normal cases, since the impedance method used by him only records the final resultant of the complex movements of the drum occurring during the same single manoeuvre.

From the above mentioned experiments it follows that the Toynbee manoeuvre is inconvenient as a testing procedure of the Eustachian tube function unless the rhinopharyngeal pressure changes are simultaneously recorded. Further, it should always be supplemented by the more exactly determinable threshold pressure applied to the closed nose and rhinopharynx. This is particularly desirable as the threshold pressure device used in this investigation always provides square wave pressure levels, adjusted in advance to the rhinopharynx, with such high accuracy during deglutitions that no further pressure control device, connected to the nose, should be needed for clinical investigations.

Owing to the threshold method described, the middle ear pressure can be indirectly controlled with the aid of the ear indication recordings. If the indication curve amplitudes are of the same size after application of both negative and overpressures of the same amount to the rhinopharynx during deglutition, the initial middle ear pressure must have been just around the ambient pressure level.

Zusammenfassung

Es werden neue Methoden für die qualitative Testung der Funktion der Eustachischen Röhre beschrieben. Dazu wurde ein Apparat entwickelt, mit welchem Druckgrenzwerte in gewünschter Höhe (Über- und Unterdrücke) innerhalb des Nasenrachenraumes während des Schluckaktes erzeugt werden können, wobei die lästige Lufteinblasung durch die Nase vermieden wird. Der Druckbereich liegt zwischen —40 und +40 mm Hg. Die Tubendurchgängig-

keit wird mit Hilfe eines Luftstrommessers festgestellt, der an den umgebenden atmosphärischen Druck angeschlossen ist. Das ganze System ist durch eine mit Glyzerin aufgefullte Gummimanschette luftdicht verbunden. Wahrend der Luftpassage durch die Tube entstehen im ausseren Gehörgang kleine Luftstosse, die als Luftstrom registriert werden konnen. Dank der hohen Empfindlichkeit des Luftstrommessers kann diese Methode sowohl für Fälle mit intaktem als auch mit perforiertem Trommelfell angewandt werden. Das Gerat ist fur klinische Tubenfunktionsprufungen gedacht und verschiedene Untersuchungen sind durchgeführt worden.

Volumetric determinations of tubal ventilation

SVEN INGELSTEDT and URBAN ORTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch M.D), University of Lund

A handy pressure chamber technique is devised for clinical studies on the Eustachian tube function A wooden box enclosing the subject to be studied functions as a pressure chamber when connected to an ordinary vacuum cleaner which can produce pressure variations from —40 to +40 mm Hg in the box. A tubing then connects hermetically the inner cuff closed part of the auditory canal of the ear to be examined with the ambient pressure outside passing airtightly through the wall of the box, i e the snorkel — a new principle The flow direction of the tubal ventilation to or from the middle ear during deglutition is then regulated by varying differently graded pressure levels in the box above or below ambient pressure

Qualitative testing of the tubal function is performed in cases with intact ear drum as well as in cases with chronic otitis by connecting a flow rate meter device to the ear snorkel tubing outside the box The determination of the volume of air passing through the tube during each deglutition presupposes a perforated ear drum (as in chronic otitis or after a drum incision) In the present investigation the volume calculations are based on determinations of the pressure changes caused by tubal air passages in a rigid closed tank of known volume, connected to a snorkel tube outside the box In this new way complete knowledge is obtained as to whether the tube can meet normal physiological demands or not since for the first time the examination is made quantitatively in both flow directions

In his monograph on the Eustachian tube, Zollner (1942) declares that investigations of the tubal function in a pressure chamber must be considered especially valuable since conditions here correspond fairly well to normal physiological circumstances Pressure chambers, however, have hitherto been available only in very few institutions and have then primarily been intended for studies of various problems concerning caisson work and aviation

However, some investigations into the function of the tube have been carried out, exposing the subjects either to excessive or moderate pressure variations Only cases with intact ear drums have so far been studied in this way It has up till now been impossible to examine cases of chronic otitis, since for natural reasons it has been impossible to establish pressure differences between rhinopharynx and middle ear in cases with drum perforations

It is the aim of the present authors to work out some form of pressure chamber method suitable for clinical purposes Further, methods are devised making possible a quantitative study of the tubal function in cases of chronic otitis Otological literature includes several studies on the tubal function, stating that the tubal function has been quantitatively determined The methods then used are either based on indirect determinations of the middle

Based on a paper read before the Swedish Otorhinolaryngological Society on May 19 1961

ear pressure by changing the pressure in the auditory canal until the pressures on each side of the drum are considered equalized, or on direct determination of the threshold overpressure in the rhinopharynx, capable of ventilating the middle ear during the opening of the tube But the amount of pressure required depends both upon the rate of air flow and the resistance offered to this flow by the tube According to the literature no such determinations have made.

Earlier investigations

In 1864 Magnus published his investigations, which had been performed in a diving bell using overpressure. He confirmed the importance of deglutition for the opening of the tube If the pressure difference between the middle ear and the bell was allowed to become too pronounced, this difference could no longer be equalized with the aid of swallowing A tubal air passage was indicated subjectively or by inspection of the ear drum

In 1872 Mach and Kessel constructed a primitive pressure chamber in the shape of a wooden box, in which pressure changes could be produced with the aid of an organ pump. The pressure ranged from —20 to +14 cm water level They made similar observations as those made by Magnus.

In 1879 Hartmann gave the classic description of the function of the Eustachian tube using a pressure chamber Among other things he described the valve-like action of the tube, allowing an easy passage of air out of the middle ear cavity, but retarding the flow back into the cavity When the overpressure in the ear exceeded a range from 20 to 40 mm Hg it was levelled out spontaneously through the tube Tubal opening for air passage was indicated subjectively or by inspection of the drum.

In 1937 Armstrong and Heim on the whole confirmed Hartmann's observations.

In 1943 Perlman made observations with the pneumophone in a pressure tank He demonstrated that accurate information about the difference in pressure on each side of the drum could be obtained with this apparatus.

In 1958 Thomsen described investigations on the tubal function by measuring the acoustic impedance of the ear in connection with pressure changes obtained in a pressure chamber Only subjects with intact ear drums could be studied and tubal air passage was indicated indirectly i e not simultaneously with the air passage through the tube

Present Investigations

Methods

Pressure Chamber

A wooden box was constructed (dimensions 200×60×60 cm) and provided with a tight-fitting lid and locking-devices The box was made of plywood, thickness 1 5 inches, and was on the inside reinforced by a hard masonite cover, thickness 0 5 inches In the lid there was an inspection window of plastic, thickness 0 4 inches. The box was furnished with a talk back system Changes in the atmospheric pressure in the box are produced by connecting it via a two-inch tubing to an ordinary vacuum cleaner (Fig 1) An overpressure is produced by turning the cleaner all around by connection The

capacity of the cleaner fan is regulated by a variable autotransformer Pressure variations from —40 to +40 mm Hg can then be produced A constant pressure level is always obtained in the box, since the fan has a comparatively great reserve capacity compensating possible moderate leakages of air The box pressure variations are controlled with a water manometer and are further recorded electrically with a pressure transducer (EMT 490 A, Elema-Schonander AB, Stockholm)

The ear snorkel system

The application of this principle means that atmospheric pressure is kept constant in the inner part of the ear canal and within the middle ear via the perforated drum of the ear to be examined, irrespectively of all pressure changes in the box The flow direction and the amount of the tubal ventilation can then be regulated during deglutition by varying differently graded pressure levels over or below ambient pressure in the box enclosing the subject This is brought about by filling a rubber cuff completely with glycerine after it has been lodged in the bony part of the ear canal in the way described by Ingelstedt and Örtegren (1963) At manometer control tests the cuff-closed space must be airtight when exposed to pressure changes from —40 to +40 mm Hg A nylon tubing, inner diameter 1 0 mm, connects the inner closed part of the ear canal with the ambient pressure since the tubing passes, airtightly mounted, through the wall of the box

Determinations of the tubal air passages

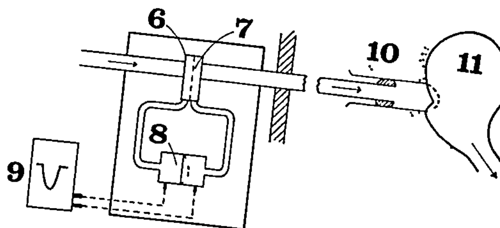
Two quite different principles have been applied

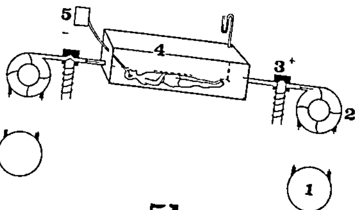
1) The qualitative testing, applicable both in cases with intact and perforated ear drum (Fig 1,5a).

Indications are made with the aid of the flow rate meter device earlier described by Ingelstedt and Ortegren (1963) This system, which opens into ambient pressure, is connected to the snorkel tube of the patient outside the box Recordings are made of the air puffs passing through the tube during middle ear ventilation and caused either indirectly by the movements of the intact drum or directly by air blowing through the perforation These indication recordings are made simultaneously with the recordings of the pressure changes within the pressure chamber on a 2 channel ECG apparatus, Mingograph (Elema-Schonander AB, Stockholm)

2) Quantitative determination of the tubal ventilation, only applicable on cases with permanent drum perforations or after incision of the drum (Fig 1,5b)

Principle If the air volume is changed within a rigid closed-tank system of known volume and kept at constant temperature, determinable pressure changes are produced in the tank From these values it is then possible to calculate every actual air mass change within the tank on the basis of the general gas law, see Appendix





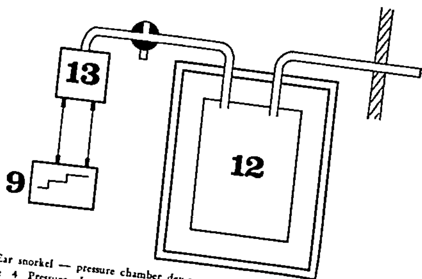


Fig 1 Ear snorkel — pressure chamber device 1 Autotransformer 2. Fan. 3 Electromagnetic valve 4 Pressure chamber 5 Flow indication devices (5a flow rate meter device 5b volume flow meter device) 6 Cylinder with pressure taps 7 Flow resistor 8 Differential pressure transducer 9 Recorder 10 Rubber cuff system 11 Middle ear 12. Rigid closed tank system 13 Pressure transducer

The experiments are carried out as follows an airtight metal tank, volume 255 ml, is connected to the snorkel tube outside the pressure chamber The tank is protected from temperature environment by being stored within an insulation box A pressure transducer, pressure range 0—30 mm Hg, is airtightly connected to the system (EMT 490 B, Elema-Schonander AB, Stockholm) Pressure changes arising in the tank and in the pressure chamber are recorded simultaneously on the ECG apparatus

During deglutition small air volumes, passing the tube, are transported to or from the tank via the drum perforation, depending on whether the air pressure in the chamber is adjusted over or below ambient pressure Owing to the sensitive pressure transducer connected to the tank, pressure measurements within the range from —1 0 to +1 0 mm Hg can be performed with high accuracy The pressure built up in the tank within this small range during the experiments cannot then seriously interfere as a counterpressure with the tubal ventilation, either qualitatively or quantitatively

The performance of the experiments

After the rubber cuff system has been airtightly lodged in the bony part of the ear canal of the ear to be examined, the patient lies down comfortably in the pressure chamber The ear is airtightly connected to the tube of the flow meter or of the closed tank system If there is a leakage of air between cuff and ear canal, this air is continuously sucked in or blown out through the flow meter, giving a simple check of the tightness of the snorkel system after application Then the sensitivity of this flow meter is so high that even a gas flow through the meter, produced by a pressure change of 1 0 mm Hg in the chamber, exceeds the working sensitivity range of the flow meter The patient swallows water on instruction by manually squeezing a water-filled compressible plastic bottle connected to his mouth by a tubing

Results

Fig 2 shows a series of ear indication (M) and chamber pressure change recordings (CH), obtained simultaneously from a normal ear case The recordings at the top illustrate how at a deglutition, when the subject is exposed to a chamber pressure of 9 cm water level below the atmospheric pressure, air passes from the middle ear through the tube and the ear drum is markedly sucked inwards when exposed to ambient pressure from outside via the snorkel system At a subsequent deglutition at overpressure in the chamber the drum bulges strongly outwards and finally, at an underpressure of 9 cm water level, reverts to its original retracted position The following recordings show the drum bulging strongly outwards when the subject is exposed to an overpressure of 6 cm water level, at the first deglutition, while the next one at the same pressure indicated by an arrow does not result in any further volume displacement of the drum Now if the chamber pressure is gradually increased

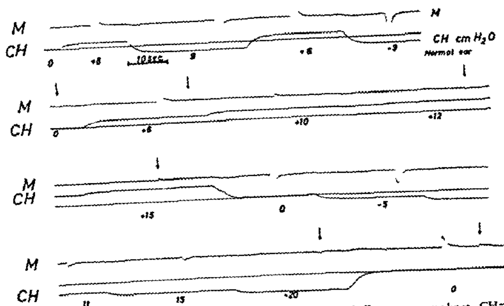


Fig 2 Flow rate meter recordings. Normal ear M=ear canal flow rate recordings. CH= chamber pressure recordings. ↓ =deglutitions.

to a 15 cm water level, the outward volume displacements of the drum during series of deglutitions are very inconsiderable The same phenomenon can be demonstrated during expositions to graded negative pressures in the chamber down to a 20 cm water level

Fig 3 shows the same experiment as in Fig 2, carried out on a case of chronic otitis with a normal tubal function Owing to the perforation, the tubal air passage can be repeatedly indicated during each deglutition at every adjusted pressure level desired in the chamber Air passes the tube at the chamber threshold pressures of —3 and +3 cm water levels

Fig 4 shows simultaneous recordings of closed-tank and chamber pressure changes in a case of chronic otitis. At a constant overpressure in the chamber of 25 cm water level, each deglutition gives rise to a tubal passage of a small air volume to the closed-tank system, giving steplike pressure variations within the range from 0 to +1 mm Hg. At increased chamber overpressures, the corresponding closed tank changes become more marked Next, corresponding tests are made at different negative pressure levels. Note the constant pressure levels recorded from the closed tank after each single act of swallowing, indicating that the system is airtight

The experiments are carried out as follows an airtight metal tank, volume 255 ml, is connected to the snorkel tube outside the pressure chamber. The tank is protected from temperature environment by being stored within an insulation box A pressure transducer, pressure range 0—30 mm Hg, is airtightly connected to the system (EMT 490 B, Elema-Schonander AB, Stockholm) Pressure changes arising in the tank and in the pressure chamber are recorded simultaneously on the ECG apparatus

During deglutition small air volumes, passing the tube, are transported to or from the tank via the drum perforation, depending on whether the air pressure in the chamber is adjusted over or below ambient pressure Owing to the sensitive pressure transducer connected to the tank, pressure measurements within the range from —1 0 to +1 0 mm Hg can be performed with high accuracy The pressure built up in the tank within this small range during the experiments cannot then seriously interfere as a counterpressure with the tubal ventilation, either qualitatively or quantitatively

The performance of the experiments

After the rubber cuff system has been airtightly lodged in the bony part of the ear canal of the ear to be examined, the patient lies down comfortably in the pressure chamber The ear is airtightly connected to the tube of the flow meter or of the closed-tank system If there is a leakage of air between cuff and ear canal, this air is continuously sucked in or blown out through the flow meter, giving a simple check of the tightness of the snorkel system after application Then the sensitivity of this flow meter is so high that even a gas flow through the meter, produced by a pressure change of 1 0 mm Hg in the chamber, exceeds the working sensitivity range of the flow meter The patient swallows water on instruction by manually squeezing a water filled compressible plastic bottle connected to his mouth by a tubing

Results

Fig 2 shows a series of ear indication (M) and chamber pressure change recordings (CH), obtained simultaneously from a normal ear case The recordings at the top illustrate how at a deglutition, when the subject is exposed to a chamber pressure of 9 cm water level below the atmospheric pressure, air passes from the middle ear through the tube and the ear drum is markedly sucked inwards when exposed to ambient pressure from outside via the snorkel system At a subsequent deglutition at overpressure in the chamber the drum bulges strongly outwards and finally, at an underpressure of 9 cm water level, reverts to its original retracted position The following recordings show the drum bulging strongly outwards when the subject is exposed to an overpressure of 6 cm water level, at the first deglutition, while the next one at the same pressure indicated by an arrow does not result in any further volume displacement of the drum Now if the chamber pressure is gradually increased

Fig 5 This pressure volume diagram is constructed from series of the preceding type of recordings as in Fig 4 The calculations are based on the general gas law On the ordinate is indicated the volume of air, in mm³ at BTPS, passing the tube during each single deglutition + means the air flow towards

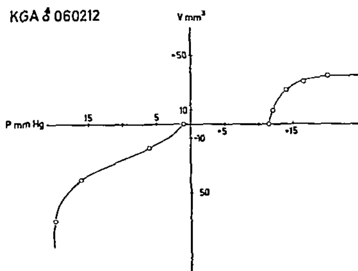


Fig 5 Pressure — volume diagram Tubal air passage starting at normal threshold pressure level at negative chamber pressure (left lower quadrant)
Tubal air passage starting at increased threshold pressure level at positive chamber pressure (upper right quadrant)

the middle ear, — from the ear to the rhinopharynx On the abscissa is given the differential pressure in mm Hg between the middle ear and the rhinopharynx, i e in practice the difference between pressure chamber and ambient pressure As seen from the figure, the tubal air passage towards the middle ear begins at a threshold pressure of 12 0 mm Hg and at the pressure of +20 mm Hg 35 mm³ of air passes through the tube during each deglutition, illustrating a reduced ventilatory function Next at the tubal air flow towards the rhinopharynx, air begins to pass at a threshold of —1 mm Hg, and at —20 mm Hg 70 mm³ of air passes the tube during each swallowing, illustrating a better function in this flow direction

Fig 6 shows the pressure volume diagrams obtained from a case of chronic otitis with a normal tubal function in both flow directions The broken line curve illustrates the original volumes passing the tube at different chamber pressures The solid line curve is obtained after repeated recordings 10 minutes after the tube had been blown through with vasoconstrictor drops Then a very marked increase of the volume flows is obtained in both directions already

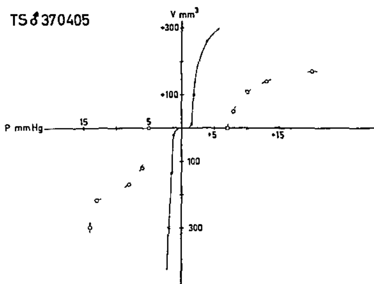


Fig 6 Pressure — volume diagram 1 O O O before and 2 X—X—X after vasoconstrictor drops applied to the tube

at minimum pressure changes within the chamber owing to the reduced swelling of the mucosa lining the tube, almost bordering on a patulous tube state

Discussion

According to Perlman (1939) and others the tubal function may be somewhat reduced when the subject is lying down as compared with the function when he is sitting up During all the actual experiments made, the subjects have been lying down since this made the construction of the pressure chamber easier and recordings of disturbing pressure fluctuations caused by involuntary movements of the subject could be reduced During the course of the investigation, however, it was shown that no disturbing pressure variations were recorded when the subject was examined outside the chamber in a sitting posture It may then be convenient to reconstruct the chamber for a sitting subject according to the principle sketched above

From the recordings in Fig 2 it appears that the drum movements following tubal air passages almost reach their maximum deflection positions inwards or outwards already when the normal subject is tested at a very moderate pressure differential between the ear and the rhinopharynx If then the chamber pressure is further varied stepwise from the ambient level, only minimum volume displacements of the drum are recorded during the tubal air passages Now, the tube has to ventilate a more or less rigid ear air chamber when the elastic properties of the tympanic membrane tend to disappear

Fig 3 shows the advantages of using the present technique when applied to a case of chronic otitis The tubal ventilation in both flow directions can be examined during a series of deglutitions giving the average function of the tube The most complete knowledge as to whether the tube can meet normal physiological demands or not, however, is obtained after volume flow determinations In the case illustrated in Fig 5 tubal air passages were demonstrated during the Toynbee experiment, which only established negative pressure changes within the rhinopharynx and the middle ear But according to the sketched pressure volume diagram the snorkel technique reveals a normal tubal ventilation only in the flow direction towards the rhinopharynx, as has been proved in the Toynbee test. An increased overpressure level in the rhinopharynx, however is needed for a middle ear insufflation This finding illustrates the fact that it is very unsafe to consider the tubal function normal in spite of a positive Toynbee test

It is not enough to estimate the degree of Eustachian tube obstruction only, it must further be proved whether the stenosis is permanent or only caused by an occasional mucosal swelling of the tube This appears from Fig 6, where vasoconstrictor drops are applied within the tube Then a marked tubal hyperventilation follows already at minimum pressure variations in the chamber

The multitude of procedures used for the treatment of chronic obstructive conditions in the Eustachian tube indicate the difficulty of the problem Investigations with the snorkel pressure chamber technique make an exact diagnosis and control of the therapeutic results possible and facilitate a selection of the most effective mode of treatment in each single case

Zusammenfassung

Es wird eine einfache Druckkammertechnik fur klinische Untersuchungen der Tubenfunktion beschrieben Eine Holzkabine fur die Versuchsperson dient als Druckkammer zu dem ein gewohnlicher Staubsauger angeschlossen wird, der Druckvariationen von —40 bis +40 mm Hg erzeugen kann Ein Schlauch system verbindet den knochernen, mit einer Gummimanschette abgedichteten Anteil des Gehorganges hermetisch mit dem umgebenden atmospharischen Druck ausserhalb der Druckkabine und kann so Messungen zuganglich gemacht werden Wir bezeichnen dieses neue Prinzip als Schnorchel Die Strom ungsrichtung der Luftpassage durch die Tube zu oder von dem Mittelohr wahrend des Schluckaktes kann durch Veranderung der Druckwerte innerhalb der Kabine reguliert werden Qualitative Untersuchungen der Tubenfunktion wurden sowohl bei Fallen mit intaktem Trommelfell als auch bei Fallen mit chronischer Otitis vorgenommen durch Anschluss eines Luftstrommessers an das Ohrschnorchelsystem* ausserhalb der Druckkabine. Die quantitative Bestimmung der Luftmenge, welche die Tube bei jedem einzelnen Schluckakt passiert setzt ein perforiertes Trommelfell voraus In der vorliegenden Unter-

suchung erfolgt die Berechnung der Luftmenge durch die Bestimmung der Druckveranderungen, die durch die Luftpassage durch die Tube in einem geschlossenen System mit bekanntem Inhalt hervorgerufen werden, wenn dieses mit einem Schnorchel ausserhalb der Druckkammer verbunden ist Auf diese Weise konnen genaue, neue Kenntnisse erworben werden ob die Tube normalen physiologischen Anforderungen entspricht, da die Untersuchungen quantitativ in beiden Richtungen vorgenommen werden konnen

CONTROLLED "EAR ASPIRATION" OF AIR

A "physiological" test of the tubal function

KNUT FLISBERG, SVEN INGELSTEDT and URBAN ÖRTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch M D), University of Lund

A new method is described, making possible a study of the tubal function following direct application of negative (or positive) pressure into the ear space. Tubal passages of air are recorded as steplike pressure variations towards equilibration when air is sucked into the closed ear manometer system during deglutition.

In ordinary conditions air passes in the aural direction through the tube when it opens during deglutition, in order to maintain the equality of pressure between the ear space and the outer air, i e "ear aspiration" During some phases in the course of an inflammatory middle ear disease, however, a negative ear pressure is very probably present because of air resorption in the ear space, provided that the tube is closed

To obtain further knowledge about the role played by the tube in normal and pathological states of the middle ear it might be of importance to supplement earlier methods used, based mainly on air inflation principles, with a new one This new method implies measuring the tubal passages of air when created by a negative middle ear pressure, while ambient pressure is kept outside the drum and within the rhinopharynx

Earlier investigations

The problem is whether it is possible, with the aid of the earlier methods used, to create "ear aspiration" of air and then to study the tubal function

1 *Intact ear drum cases*

a) Pressure chamber

The whole subject must first be exposed to negative pressure, to which the ear spaces should be equalized by swallowing After this procedure the negative pressure of the chamber must be brought back again to the ambient pressure At this point the function of the tube can be studied because air is sucked into the ear during swallowing, provided that the tube function is normal This is a wellknown phenomenon in aviation This test method has been used by several authors but is not suitable for clinical use

b) Pneumophone method (v Dishoeck)

Under certain conditions it is possible with the aid of this device to suck out the ear drum by a negative pressure applied to the ear canal, thereby

Based on a paper read before the Swedish Otorhinolaryngological Society on December 2 1961

creating a negative middle ear pressure The ability of the subject to equalize this negative pressure by swallowing is then an expression of the tube function
c) The Toynbee manoeuvre under certain conditions

In cases where this manoeuvre results in a remaining negative middle ear pressure, the ability of the tube to equalize this negative pressure during ordinary swallowing can be studied by means of different indication methods

2 *Perforated ear drum cases*

Curiously enough, there has up till now been no available method of performing "ear aspiration" studies of the tubal function in cases of chronic otitis The present authors have therefore worked out a method suitable for clinical use The principle of the method is based on the "reversed" ear snorkel — pressure chamber technique

Method

A prerequisite for using this method is that free air contact between the ear canal and the middle ear space already exists, i e a drum perforation is permanent or can be created by a drum incision (Fig 1) If a closed manometer system is airtightly connected to the ear space, the whole combined system will go on being closed as long as the subject does not swallow Pressure variations within the ear are then easily produced by the investigator in the closed

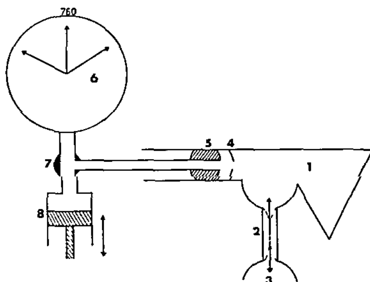


Fig 1 Principle of "aspiration method 1) Air filled ear space 2) Eustachian tube 3) Rhino pharynx 4) Ear drum 5) Rubber cuff 6) Manometer 7) 3 way stopcock 8) Syringe

system outside the ear Afterwards the system may be opened by the subject himself during swallowing and air then passes through the tube Different degrees of pressure decrease towards ambient pressure within the closed system can be recorded

Two manometer systems have been used alternatively, depending on the pressure range within which it has been necessary, in the individual case, to investigate the tubal function (pressure transducers EMT 490 A, pressure range 0—300 mm Hg and EMT 490 B, pressure range 0—30 mm Hg, Elema-Schonander AB, Stockholm) The manometer is connected with a nylon tubing to the cuff closed ear space and the manometer is provided with a 3 way stopcock for producing necessary pressure variations within the total closed ear manometer system After being transformed and amplified the transducer signals are recorded on a Mingograph

The pressure levels used in the closed system before studying the tube function have the following ranges —2, —5, —10, —20, —30, —40 and —50 mm Hg During recordings in the course of deglutition the degree of pressure reduction in the closed system then provides information on the tubal function Owing to the low sensitivity range of the transducer required for recording within the pressure range, disturbing pressure fluctuations due to movements of the subject himself are not recorded The subject can therefore be allowed to sit up in a chair with a headrest Furthermore, thanks to the short time needed for each study there is no time for disturbing thermal influences on the enclosed manometer gas

Example of a closed ear manometer recording

In Fig 2, after application of —10 mm Hg within the middle ear on a normal ear case after a drum incision, the recorded negative pressure level is

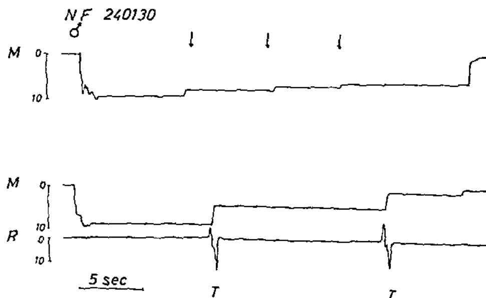


Fig 2 Recordings from a normal ear case M=pressure change recordings in mm Hg in the closed ear space at tubal air passage ↓ ="open nose deglutition. T=Toynbee manoeuvre. R=rhinopharyngeal pressure changes.

stepwise reduced indicating that air passes into the ear simultaneously with each deglutition (upper curve)

The lower curves show the pressure changes made by "aspiration" + inflation during Toynbee on the same case Air enters the middle ear only during the positive phases

Comments

It is thus possible, with this method, to determine at which level of negative or positive pressure applied within the ear space air passes through the tube during swallowing Thus if the volume of the closed ear-manometer system could be determined, every volume of air passing through the tube could be quantitatively determined with the aid of the general gas law (see Appendix) owing to the pressure change produced in the closed system The solution of this appears from the following papers

Zusammenfassung

Es wird eine neue Methode beschrieben, die eine Untersuchung der Tubenfunktion durch direkten Anschluss von negativem (oder positivem) Druck an das pneumatische System des Mittelohres ermoglicht Die Luftpassage durch die Tube wird als schrittweise Druckanderung registriert, die innerhalb des geschlossenen Ohrmanometersystems bei jedem Schluckakt erfolgt

CLINICAL VOLUME DETERMINATION OF THE AIR-FILLED EAR SPACE

KNUT FLISBERG SVEN INGELSTEDT and URBAN ORTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch, M.D),
University of Lund

A new method for exact clinical volume determination of the air filled ear system is devised. The method is based on the general gas law A volume variation between 0 to 25 ml is obtained from 85 cases

Several studies have been made on the size and distribution of the air cell system of the human ear, since variations in pneumatization are of importance in ear pathology According to Diamant (1940) different types of pneumatization depend on a genetic variation, while Wittmaack (1918) and Tumarkin (1957) hold a pathologic ear process to be the cause of poor cell formation The function of the air cell system must still be considered unclear Link and Handl (1955), however, have reported a high incidence of noise deafness in subjects with poorly pneumatized ears, but Diamant (1962) could not confirm such a relation

The size of the air cell system can only indirectly be estimated with the aid of clinical X ray examination, but so far it has only been possible to make direct volume determinations of the air space on sectioned temporal bones

Earlier investigations

Roentgen planimetry for determination of the distribution of the air cell system has been performed by, among others, Kurata (1938), Diamant (1940), and Tumarkin (1957) The distribution area of the air cell system was measured on films when exposed in some lateral head position Areas ranging from 0 to 30 cm² are reported However, these were always very small (0—3 cm²) in cases with chronic otitis

In 1950 Silbiger reported direct volume determinations on sectioned temporal bones of 200 normal ears. The air spaces of the preparations were filled with water by a special vacuum technique. The water volumes then obtained in the air cell system varied from 6 to 35 ml.

Determinations of the volume of the air-filled space of the ear is a prerequisite for further studies of the function of the middle ear and the Eustachian tube Therefore, the present authors have worked out a clinical method for direct volume determination of the air space of the ear.

Based on a paper read before the Swedish Otorhinolaryngological Society on December 2, 1961

Method

Principle The method is based on the general gas law This means that for a given mass of the same gas $\frac{P \cdot V}{T}$ is constant (P=pressure, V=volume, T=temperature) If the temperature is kept constant Boyle's law is applicable where $P_1 \cdot V_1 = P_2 \cdot V_2$ In Fig 1 the principle is illustrated It

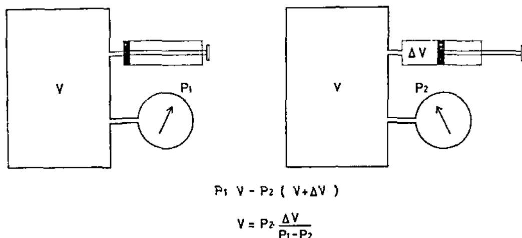


Fig 1 The principle of air volume determination

appears from this that when the volume increases by displacing the piston outwards, the gas density diminishes and the pressure in the closed system falls If the original pressure and the actual changes of volume and pressure are determined, the original system volume can be calculated from Boyle's law

The mode of application appears from Fig 2 In the bony part of the auditory canal the cuff is airtightly inflated with glycerine according to the method described by Ingelstedt and Ortegren (1963) The inner part of the ear canal is then connected to an electromanometer, pressure range 0 to 30 mm Hg, type 490 B (Elema-Schonander AB, Stockholm) The output of the manometer is amplified and fed into an ECG apparatus The measuring system is provided with a pipet device filled with water by which known volume changes of gas can be produced by displacing the water level with an accuracy of 0 5 mm^3

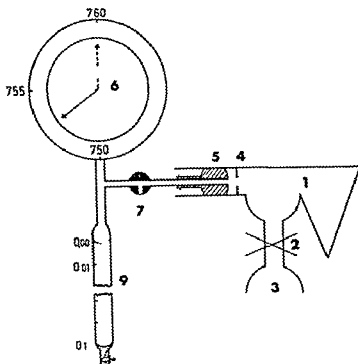


Fig 2 Application of volume determination device 1) Air filled ear space 2) Eustachian tube 3) Rhinopharynx 4) Ear drum 5) Rubber cuff 6) Manometer 7) 3 way stopcock 9) Pipet

Performance

The following air volumes are determined

1) Manometer system + the volume of the ear canal space between the cuff and the intact ear drum.

2) Manometer system + ear canal + airfilled ear space, supposing that an original perforation is present or an incision is made in the intact drum

For the calculation of the volume of the air cell system only, the volume of the measuring system + the enclosed ear canal space up to a normal ear drum must first be measured.

The cuff is lodged in the bony part of the ear canal If this is done repeatedly, the cuff cannot be placed at exactly the same site Series applications on the same subject for six days showed volume variations ranging from 0 96 to 1 28 ml of the closed air space between the cuff and the intact drum. The corresponding volume variations of 50 normal ears in different subjects after one application ranged from 0 52 to 2 12 ml, mean 1 1 ml This mean of 1 1 ml is called the "standard volume" of the ear canal.

The volume of the measuring system has been kept constant during all the experiments and has been controlled regularly The volume variations at

different determinations ranged from 3 45 to 3 55 ml, mean 3 50 ±0 05 ml This illustrates the accuracy of the method

When calculating the volume of the ear space only, in the case of an original drum perforation, the volume of the measuring system and the "standard volume" of the ear canal (i e 4 6 ml) must be subtracted from the total ear manometer volume obtained

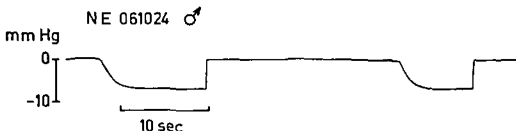


Fig 3 The air volume increase by pipet suction causes a simultaneous pressure change recorded within the closed system

In Fig 3 the pressure fall in the closed ear manometer system is recorded caused by a volume increase of 0 1 ml The total volume in this case was determined at 10 8 ml

The patient must not swallow during any pressure recordings, thereby preventing air passage through the Eustachian tube

Results and Discussion

Volume determinations have been made on 85 ears — normals and cases of chronic otitis The volumes obtained have varied from 0 to 25 ml

Roentgenograms are indispensable as an aid to show the cellular distribution and the type of air cells within the temporal bone However, the absolute size of the total air space as a functional unit can only be measured by a volumetric method Exact volume values are needed for further studies on the function of the middle ear and of the Eustachian tube, as will appear from the following papers

Zusammenfassung

Es wird eine neue Methode fur genaue klinische Volumenbestimmung des pneumatischen Systems des Mittelohres beschrieben Als Grundlage fur die Berechnungen dient die Anwendung des Idealen Gasgesetzes Bei 85 untersuchten Fallen konnten Volumenunterschiede von 0 bis 25 ml festgestellt werden

ON MIDDLE EAR PRESSURE

KNUT FLISBERG, SVEN INGELSTEDT and URBAN ORTEGREN
Lund, Sweden

*From the Department of Otolaryngology (Head Prof Hj Koch, M D),
University of Lund*

A new method for producing pressure variations in the pneumatic system of the human ear under control is described, i.e the mastoid puncture technique Several investigations are made, the volume displacement capacity of the normal ear drum, the role of the drum as a middle ear pressure regulator, vacuum effects on hearing and transudation, all these changes are produced by intra aural pressure variations under control. The accuracy of the previous methods for indirect determination of the middle ear pressure is analysed

It is a well-known fact that the middle ear pressure must be kept equal to the ambient pressure level for keeping a normal middle and inner ear function If for some reason the middle ear ventilation through the tube should disappear, a negative pressure is built up, caused by gas diffusion into the blood of the mucosal vascular bed In order to investigate this phenomenon, various attempts have been made to measure the middle ear pressure. In man, however, only methods by which this pressure is indirectly measured have been used so far and the results obtained seem to have been inaccurate, at least under certain experimental conditions Since it is scientifically and clinically important to find a direct method of determining the middle ear pressure and simultaneously of changing it under control, new methods have been worked out

Earlier Investigations

Direct methods

Such experiments have been performed by, among others, G Holmgren (1934) and Handl (1957) By puncturing the bony wall, these authors created a direct communication with the ear space on animals L. Holmgren (1940) tried to measure the middle ear pressure by means of ear drum puncture. Animal experiments have been performed by Wever, Bray and Lawrence (1942) and by Wever, Lawrence and Smith (1948) Metz (1946) measured the changes in the impedance of the drum following middle ear pressure variations on temporal bone preparations

Indirect methods

These methods are based on the assumption that when a pressure equilibration state is produced between the closed spaces of the external ear canal

Based on a paper read before the Swedish Otorhinolaryngological Society on December 2, 1961.

and that of the middle ear owing to the mobility of the drum, both pressures are equal. The pressure difference between the atmosphere and the ear canal should then be equal to the pressure difference between the atmosphere and the middle ear cavity. The state of pressure equilibration has then been determined in different ways.

1) *Optically*, i.e. by the manometer method according to Krassnig—Zollner. During microscopic control the drum is forced by the external pressure change into its "normal position", i.e. the position taken by the drum when the ear pressure is equilibrated to ambient pressure.

2) *Acoustically*.

a) The pneumophone method (v. Dishoeck).

This author considers the pressures on both sides of the drum to have been made equal when a constant tone, simultaneously sent into the ear canal, is heard most distinctly by the subject.

b) The impedance measuring method when used for tubal function studies by Thomsen (1957) in a pressure chamber. The pressure outside the drum, i.e. the chamber pressure, is changed and the state of pressure equilibration was supposed to have been reached at the point when the maximum absorption of a pure tone was registered.

Zollner, however, has pointed out that certain sources of error are involved when methods are based on the above mentioned principle. If the air volume of the closed ear space should be small owing e.g. to bad pneumatization, to undiagnosed presence of fluid within the ear, or to a large, thin, and mobile scar on the ear drum, then the pressure equilibration procedure itself will seriously influence the middle ear pressure to be determined. This is due to the fact that a volume displacement takes place owing to the mobility of the drum. According to Boyle's law the actual pressure within the closed ear space is then directly related to the volume displacement produced. Zollner, however, has not made any determinations of these pressure volume relations, so the magnitude of this possible error is so far unknown since no later investigators have paid any attention to his good suggestions.

Present Investigations

The sketch of Fig. 1 shows the factors that regulate the middle ear pressure and some disturbances of the normal middle ear function caused by direct application of negative pressure within the ear space. It must be observed, however, that problems of gas diffusion to or from the ear space are being investigated. These problems, however, offer many difficulties from both methodological and physiological points of view.

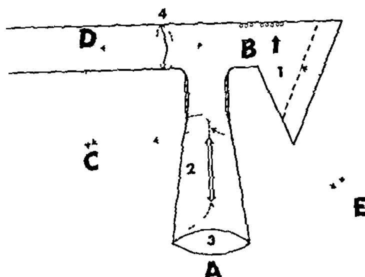


Fig 1

Factors regulating the middle ear pressure

Main factors

Tubal ventilation (A)
Gas diffusion (B)
Ear drum mobility (D)

Vacuum effects (C) on
hearing
transudation
tubal locking

Accessory factors (E)

Variation in air space volume
Tubal factors
secretion
surface tension
ciliary activity

(1 Air filled ear space
2 Eustachian tube
3 Rhinopharynx
4 Ear drum)

The Direct Puncture Technique of the Air Cell System

Studies according to the principles sketched in Fig 1 require an intact ear drum Further a catheter cannot be introduced into the tube via its rhinopharyngeal orifice since this would seriously disturb the ventilatory capacity of the tube Another drawback is that such a procedure causes discomfort to the patients which makes further investigations difficult The only remaining way of obtaining a direct communication with the air filled ear space is then to puncture an air cell at the tip of the mastoid First, of course, the direction and the size of the air cell system within the mastoid process must be determined by roentgenogram

Procedure

The skin covering the process is anesthetized with 1 % xylocain. The tip cell is then punctured with a straight mandrin needle through the cortical bone as appears from Fig 2 In most cases this can be performed without any bleeding into the air cells which is a prerequisite for obtaining accurate

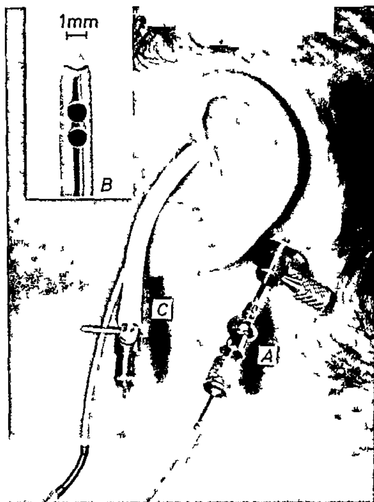


Fig 2
Puncture needle arrangements
A Puncture needle
B Detail of needle
C *Cuff system lodged in ear canal*

pressure change recordings from the ear space Even the smallest drop of fluid will seriously disturb the recordings by blocking the lumen of the needle or the air passage from the needle to the middle ear cavity If by any chance such a disturbance should occur it is easily recognised on the curves

Pressure changes within the closed ear space are then made via the needle It must be expected that the negative pressure variations are of greater interest than the positive ones, because they are regarded as common in inflammatory middle ear diseases A negative pressure application must be regarded as harmless on condition that no extreme negative pressure is applied in the closed ear space An overpressure, on the contrary, should be applied very carefully under thorough and continuous controls, making sure that the airway through

the system is really free There is a risk that unskilful application of over-pressure may involve puncture of a vein, which might result in air embolism

By this technique about twenty ears have so far been punctured without any complications either in connection with the investigations themselves or afterwards Nor have the puncture and the subsequent investigations been unpleasant to the patients It has been possible to carry out a number of different examinations of the ear by this puncture technique.

1 *Volumetric Determinations of the Closed Air-filled Ear Space and of Tubal Ventilation.*

These determinations have been made according to the principles earlier described The closed manometer system (EMT 490 B, pressure range 0—30 mm Hg, volume displacement 0 07 mm^3/1 mm Hg) has been connected to the puncture cannula airtightly passing through the cortical bone of the mastoid For the results obtained see the previous paper

2 *The Degree of Drum Excursions when Determined as Volume Displacement, following Controlled Middle Ear Pressure Variations.*

Apparatus, procedure and calculations

A closed manometer system, (EMT 490 B, pressure range 0—30 mm Hg, displaced volume 0 07 mm^3/1 mm Hg) is airtightly connected to the bony part of the ear canal, after which the total closed air volume of the system is determined up to the intact ear drum according to the principles earlier described Another manometer system (EMT 490 A, pressure range 0—300 mm Hg, displaced volume 0 07 mm^3/100 mm Hg) is then directly connected to the mastoid puncture cannula A 20 ml syringe can be connected to the system via a 3-way stopcock mounted between the manometer and the cannula. With the aid of this syringe air pressure variations can be produced within the closed ear cannula manometer system, see Fig. 3

Pressure changes caused by drum movements are then recorded in the closed ear canal manometer system, when the intra-aural pressure is varied via the cannula Both pressure curves are simultaneously recorded

From the pressure changes, caused by the drum movements, the degree of drum excursions can be calculated as a volume displacement in mm^3 counted from its "resting position The calculations needed are based on the general gas law In the case recorded on Fig 5, for example, the closed manometer system used for pressure recording within the ear canal has an air volume of 6 3 ml up to the intact drum. Now if the pressure values obtained in the system are directly used for the volume displacement calculations, an approximation only is made Then simultaneously with the movement of the drum, i e resulting in a volume displacement, a counterpressure is built up within the

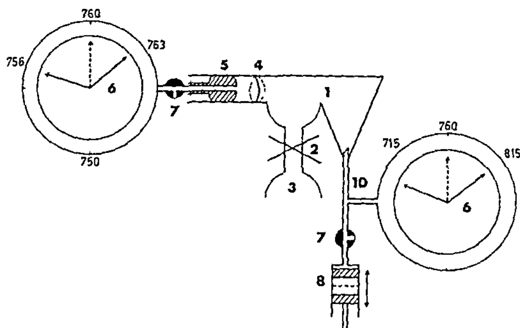


Fig 3.

Sketch of drum excursion determination arrangements

1) Air-filled ear space
2) Eustachian tube
3) Rhinopharynx
4) Ear drum
5) Rubber cuff
6) Manometer
7) 3-way stopcock
8) Syringe
10) Puncture needle

closed ear canal-manometer system. Hence in working out the diagram between volume displacement and pressure, the pressure difference across the drum should be used, after which the ear canal pressure is reduced to ambient pressure. See Table I, and Fig. 6.

TABLE I

Recorded values			Corrected values
Middle ear pressure in mm Hg	Ear canal pressure in mm Hg	Drum volume displacement in mm^3	Drum volume displacement calculated at 760 mm Hg in mm^3
1 33	0 67	6 0	6 2
4 67	1 08	8 0	8 6
5 00	1 25	10 0	10 5
6 67	1 41	11 9	12 0
8 67	2 00	16 5	17 0
16 67	2 50	21 0	21 0
20 00	3 33	28 0	28 0
40 00	4 00	34 0	34 0
50 00	4 17	35 0	35 0

The pressure changes must further produce some volume displacement by loading the membrane of the manometer itself The volume displaced for the manometer used is *0 07 mm³/1 mm Hg* This volume displacement occurring within the manometer system is negligible when the above mentioned calculations are made in none of the cases studied did the ear canal manometer pressures caused by the drum movements exceed 4 2 mm Hg

Results and Comments

In Fig 4 the maximal pressure changes in the closed ear canal manometer system are recorded when the normal ear drum is forced outwards and inwards *by rapid positive and negative intra aural excess pressure variations,* and both pressure curves are recorded simultaneously Four normal middle ear cases have *been studied in this way When the maximal volume displacements outwards* and inwards, respectively, have been calculated as volume deviations from the

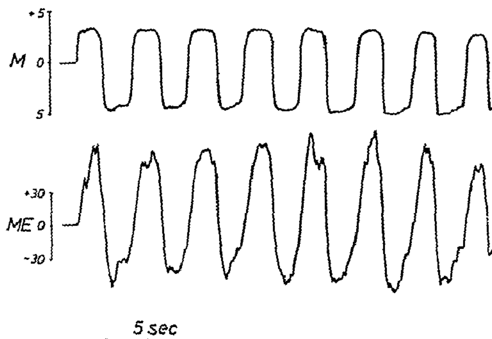


Fig 4
Simultaneous pressure change recordings outside and inside the ear drum
M=ear canal pressure recordings
ME=middle ear pressure recordings.



Fig 5
Ear canal pressure variations caused by normal drum movements at steplike lowered middle ear pressure in mm Hg

"drum resting position in mm³, both ranged from 25 to 40 mm³ In Fig 5 the pressure changes in the closed ear canal manometer system, caused by the varied degrees of movements of the normal ear drum, are recorded when produced by varied steplike pressure variations made in the closed ear space below and above ambient pressure level From these recordings the relationship between the intra aural pressure change and the ear drum volume displacement has been calculated and the values appear in Fig 6

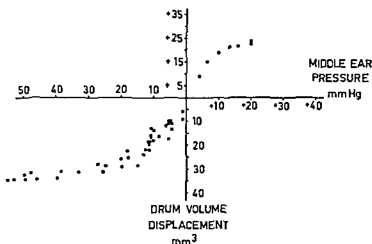


Fig 6
Calculated pressure — volume displacement relat on

The ability of the normal drum to regulate the middle ear pressure by volume displacement appears from the figure Thus, 60 per cent of the drum's maximal capacity for volume displacement, when counted from a "resting position", has already been elicited by a middle ear pressure of —10 mm Hg Every further lowering of the pressure in the closed ear space will from now on be applied in an ear space which more and more assumes the character of a rigid chamber

The results of these investigations give us a possibility of estimating the accuracy of the above mentioned indirect methods of determining middle ear pressure, since the relation between the pressures inside and outside the normal drum under the actual experimental conditions can be read from Fig 7

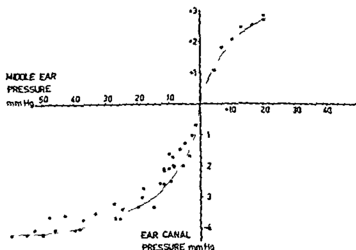


Fig 7
Recorded middle ear — ear canal pressure relation.

Fig 8a illustrates what really happens when the gas volume Δ v is evacuated from the known ear space air volume V at atmospheric pressure The new situation is characterised by a volume decrease V_3 owing to the inward displacement of the drum This displacement in its turn can be calculated with the aid of the general gas law from the reduction of ear canal pressure The reduced pressure in the middle ear P_1 can be read directly After the observations made, the following questions can be answered how great is the error in the determination of the middle ear pressure in the indirect procedure of measur-

ing the pressure in the ear canal after compensation of a volume displacement of the drum, and how great was the volume Δ v evacuated from the middle ear during the procedure?

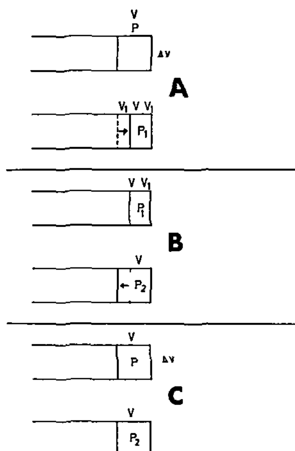


Fig 8
For details see text

Fig 8b illustrates what happens at measurements with e g the pneumophone method P_1 is the pressure required at the ear space volume of V-V_1 Now if this volume, just as at pneumophone determination, is brought back to its original volume V, the pressure P_2 is obtained and can be calculated according to Boyle's law $= (V\ V_1)\ P_1 = V\ P_2$ The measurement error resulting from the use of an indirect pressure determination procedure can be explained as follows no attention has been paid to the fact that a volume displacement has changed the pressure from P_1 to P_2 owing to the mobility of the drum, as appears from Table II

At C in Fig 8 the gas volume Δ v, evacuated from the closed ear space, *can be calculated from the general gas law when* $\Delta\ v = \frac{P-P_2}{P}\ V$, *see Appen*-dix and Table II

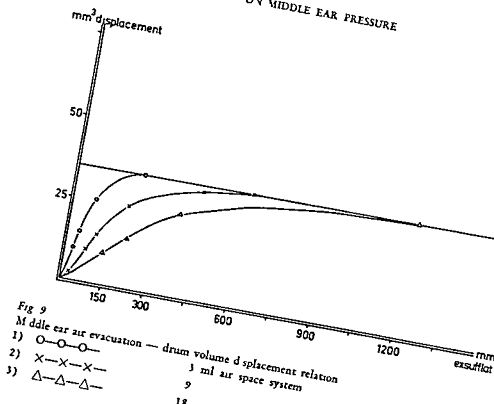


Fig 9

Middle ear air evacuation — drum volume displacement relation

1) O—O—O— 3 ml air space system

2) X—X—X— 9

3) Δ—Δ—Δ— 18

Fig 9 shows the degree of the drum volume displacement related to Δ v according to the above principles at three different sized air volumes of the ear spaces

Conclusions

The volume displacement caused by the normal ear drum at varied directly controlled middle ear pressure variations can be calculated The role of the ear drum as a regulator of the middle ear pressure is analysed and shown in Table II in the column P_2—P_1 (drum factor) where P_2 refers to the middle ear pressure, supposing the ear space to be a rigid chamber, and P_1 expresses the pressure values obtained under the same conditions when the ear space is provided with a normal ear drum Thus assuming that a given air volume is evacuated from ear spaces of different sizes the role of the drum, when functioning as a middle ear pressure regulator, becomes greater the smaller is the air volume of the ear space (Fig 9) The accuracy of the indirect middle ear pressure determinations is analysed It appears from this that indirect methods for quantitative middle ear pressure determinations can only be used if simultaneous determinations are made of the air volume of the closed ear space and of the degree of volume displacement of the drum Otherwise

measurement errors may occur which increase both with decreasing ear space volume and with an increasing mobility of the drum These factors are demonstrated in Table II

TABLE II

V ear space volume in mm³	v air volume evacuated in mm³	V_1 drum volume displacement by v in mm³	$P\ P_1$ middle ear pressure change by v in mm Hg	$P\ P_2$ middle ear pressure calculated at V in mm Hg	$P_2\ P_1$ drum factor in mm Hg	$\frac{P_2\ P_1\ 100}{P\ P_1}$ the error by using pressure equilibration methods
3000	10 6	2 5	2 0	2 7	0 7	35 0
	17 0	5 0	3 0	4 3	1 3	43 0
	31 6	10 0	5 0	8 0	3 0	60 0
	43 4	15 0	7 5	11 0	3 5	47 0
	83 0	25 0	15 0	21 0	6 0	40 0
	127 0	30 0	25 0	32 0	7 0	31 0
	170 0	32 5	35 0	43 0	8 0	23 0
	230 0	35 0	50 0	58 3	8 3	16 5
9000	26 0	2 5	2 0	2 2	0 2	10 0
	41 5	5 0	3 0	3 5	0 5	16 5
	71 0	10 0	5 0	6 0	1 0	20 0
	107 0	15 0	7 5	9 0	1 5	20 0
	207 0	25 0	15 0	17 5	2 5	16 5
	319 0	30 0	25 0	27 0	2 0	8 0
	446 0	32 5	35 0	37 7	2 5	7 1
	626 0	35 0	50 0	53 0	3 0	6 0
18000	49 7	2 5	2 0	2 1	0 1	5 0
	73 9	5 0	3 0	3 1	0 1	3 4
	130 0	10 0	5 0	5 5	0 5	10 0
	240 0	15 0	7 5	9 5	1 0	13 5
	380 0	25 0	15 0	16 0	1 0	6 6
	616 0	30 0	25 0	26 0	1 0	4 0
	872 0	32 5	35 0	36 6	1 6	4 6
	1231 0	35 0	50 0	52 0	2 0	4 0

3. *Negative Middle Ear Pressure Effects upon Hearing*

An audiogram is performed at varied negative pressure levels applied within the normal ear space via the mastoid puncture cannula

Results and Comments

Only few experiments have dealt with this problem but no investigations seem to have been performed during direct control of the middle ear pressure in man A change of the pressure in the middle ear cavity may be supposed to influence the hearing both by a change of the sound transmission through the middle ear, resulting in a threshold rise more pronounced for air conducted than for bone conducted sounds, and by an impaired transmission through the inner ear This leads to a threshold rise also for bone conducted sounds These well-known hypotheses have been verified by testing, see Fig 10 At

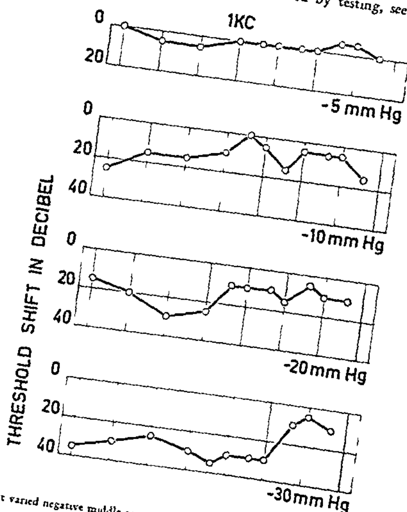


Fig 10
Hearing loss at varied negative middle ear pressure

small negative pressures in the middle ear the transmission of low tones is first impaired, and, at decreased pressures, also the transmission of high tones is affected Still greater pressure decrease even affects the bone conduction

This investigation has been made in collaboration with Dr H E. Holst.

4 *Hydrops ex Vacuo in the Human Ear*

"We do not know how long the tube remains closed before pressure drops or fluid appears in the middle ear" (Perlman 1960)

This type of studies should naturally only be performed in cases of Meniere's disease, which lend themselves to operative destruction of the labyrinth

With the aid of the mastoid puncture cannula, varied negative pressure levels are applied to the closed ear space in four cases

Results

Transudation can be produced in the ear on the following conditions

Amount of negative pressure applied into the ear in mm Hg	Time needed for demonstrating transudation in minutes
20 to 30	15
100	5

The transudation consists of a thin clear yellow fluid, which can be sucked out of the ear via the mastoid puncture cannula

Zusammenfassung

Es wird eine neue Methode beschrieben fur die Erzeugung von kontrollierbaren Druckvariationen im pneumatischen System durch die Punktion des Prozessus mastoideus Folgende Untersuchungen wurden vorgenommen die Bewegungskapazitat des normalen Trommelfells, die Bedeutung des Trommelfells als Druckregulator im Mittelohr, die Wirkung von Vakuum auf das Horen und die Transsudation Alle Veranderungen wurden bei kontrollierten intratympanalen Druckanderungen untersucht Die Genauigkeit fruherer Methoden fur indirekte Bestimmung des Mittelohrdruckes wird analysiert

THE VALVE AND "LOCKING" MECHANISMS OF THE EUSTACHIAN TUBE

KNUT FLISBERG, SVEN INGELSTEDT and URBAN ÖRTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head: Prof. H. Koch, M.D.), University of Lund

In controlled "ear aspiration" experiments on normals it is shown that the air volumes passing through the tube during deglutition gradually decrease with lowering of the negative middle ear pressure. If tympanic inflations are performed simultaneously, i.e. "aspiration + inflation with a very slight overpressure, the air volumes passing the tube at identical middle ear pressures are far greater as compared with "aspiration" only.

At "common cold" the tube can be "locked" by very small negative middle ear pressure. Now, if during the "locking" period attempts are made to open the tube by swallowing, further negative pressure spikes are simultaneously produced in the closed ear space thanks to the action of the muscular opening forces on the tube. In this way these forces oppose themselves. These phenomena are further illustrated by an ear Eustachian tube model.

The normal Eustachian tube is easily forced open by an overpressure in the tympanic cavity (Hartmann 1879). When the extra tympanic pressure increases, however, the tube behaves in an entirely different manner: then the tube does not open passively and no air enters the middle ear without intervention of muscular action or by active inflation. If this overpressure outside the ear reaches a level of about 90 mm Hg the tube is "locked". It is now impossible for the tube muscles to overcome this great pressure difference since the membrano-cartilaginous portion has collapsed tightly (Hartmann, Armstrong and Heim [1937] and others). McGibbon (1942) illustrated the mechanism of this type of "locking" with the aid of an ear-Eustachian tube model as a purely mechanical "pressure occlusion". As a secondary effect, a "vital occlusion" would then take place owing to lesions of the mucous membranes in the tube.

The authors question the possibility that the Eustachian tube may be closed to "ear aspiration" during ordinary swallowing and, *in spite of this, open to* inflation. This was in fact suggested by L. P. Fowler in 1920. Then perhaps a successful inflation might be inaccurate as a functional measure of the lumen patency.

Earlier Investigations

For comprehensive reviews of the anatomy and physiology of the tube, see Zollner (1942), Graves and Edwards (1944), Perlman (1951), and Aschan (1955).

It is obvious from several investigations that the normal tube, when there is only a small or no pressure differential at all between the ear space and the ambient pressure, only opens occasionally during swallowing. This is assumed

to be due to different degrees of separation of its walls (Gyergyay 1932, Zollner and Perlman) Already in 1869 Cleland suggested that the mucous film causes the closure of the narrow part of the tube Further, Perlman suggested that the lumen opening of the tube may be related to the breaking up of the mucous blanket Aschan made radiological studies of the tubal function during swallowing in cases of chronic otitis and could nicely distinguish several phases in the mechanism of the tubal function by combining his anatomical and radiological findings Thus, the tube is open during a very short phase, and the active muscular opening starts at the pharyngeal end The lumen closure, starting from the aural end, is due to relaxation of the muscles supported by elastic fibres in the tubal cartilage and the capillary forces between the damp surfaces of the mucous membranes of the tube Aschan also found that the tube did not always open during swallowing

Now a further question arises can pressure changes be produced in the closed ear space during the opening phase of the tube, before any direct air connection has been established between ear and rhinopharynx during swallowing?

Zollner describes pressure changes in the ear which are "brought about by the movements of the tube itself" Similar observations have been made by Perlman, who suggests that the separation of the walls of the tube by abrupt breaking up of the mucous blanket may explain the initial movement of the ear drum during swallowing "The mucous blanket break suddenly increases the active volume behind the drum, thus reducing its pressure and causing an inward movement of the drum"

Surface Tension

The part played by the surface tension in the tubal mechanism has been indicated by several authors, but none of them have dealt with the problem For elucidation of the basic problems of surface tension we should in the first place turn to works on lung physiology, since similar problems are met there

Every fluid tends to form the least possible surface, and at the interface with air, the molecules at the surface are attracted more strongly to their neighbours below the surface and are only weakly attracted to the sparser population of molecules in the air above the surface The smaller radius of curvature, the greater will be the pressure difference required to maintain the curvature

In 1929 von Neergaard was the first to demonstrate the contribution of surface tension to the elasticity of the lungs Later on these investigations have been developed by Mead, Whittenberger and Radford (1957) If lung preparations are alternatively distended with air and with saline solution, and if the pressures required are compared, it takes a much higher pressure to distend the lungs from their air-free state with air than it takes to do it with fluid. During emptying, however, the air- and saline-filled lungs had both about

the same pressure volume characteristics "As air is forced along successively smaller branches of the bronchial tree during inflation from air-free state, pressures opposing surface tension must increase" (Mead *et al*) The present authors are of the opinion that these observations are also applicable in the case of the Eustachian tube

PRESENT INVESTIGATIONS

1 "*Ear Aspiration*"-*Inflation*

Comparative quantitative determination of the middle ear ventilation through the tube during swallowing, either caused by a negative pressure in the ear, i e "aspiration", or by an overpressure in the rhinopharynx, i e inflation, or finally when caused by a combination of "aspiration" inflation

Procedure

The constant overpressure or negative pressure required when applied in the closed ear space to produce tubal air passages can be obtained either through the mastoid puncture or through a drum incision According to principles described earlier, a closed manometer system is then airtightly connected to the ear space Next the total manometer ear space volume is determined If a negative pressure is now produced in this closed system, different degrees of pressure fall to atmospheric can be recorded when air passes through the tube during swallowing and each volume of air entering the closed ear manometer space can be calculated Inflation studies have only been performed on such subjects as regularly produce positive pressure in the closed rhinopharynx during Toynbee manoeuvres and equally regularly open their tubes during these positive pressure pulses Thus no greater variation of the inflation pressure has been produced in the individual case However, in these initial comparative studies the authors have intentionally avoided producing inflation by means of unphysiological methods During the Toynbee tests the pressure variations in the ear space and in the rhinopharynx have been recorded simultaneously

Results and Comments

In Fig 1 the relationship between the middle ear pressure and the volume of gas entering the middle ear is illustrated in a normal case The curve marked ●—●—● represents this pressure gas exchange relation following "aspiration" only, and the curve O—O—O represents the same relation following "aspiration" + inflation, when inflation is produced by a positive pressure of 5 mm Hg during Toynbee It may be observed that much greater volumes of air enter the ear on "aspiration" + inflation at a smaller negative middle ear

pressure than the corresponding volumes produced by "aspiration" only As judged from both these volume curves, the tubal ventilation during swallowing is more than three times greater after "aspiration" + inflation (+5 mm Hg) than after "aspiration" only within the middle ear pressure range from —20 *to —30 mm Hg*

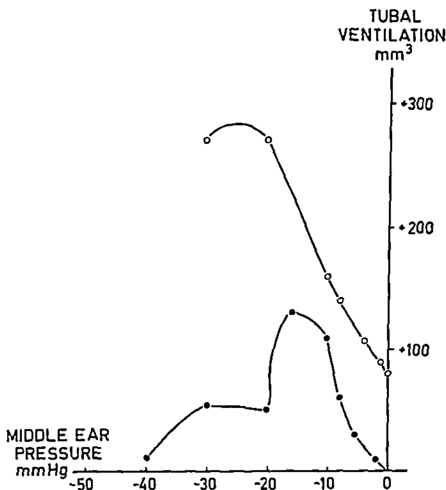


Fig 1
Tubal air passages by "aspiration and "aspiration" + inflation
● — ● "aspiration"
O — O " " "+inflation (+5 mm Hg)

In Fig 2 from another normal ear case, the pressure-volume curves show similar tendencies as in case one Here the inflation is produced by a positive pressure of +1 mm Hg during Toynbee

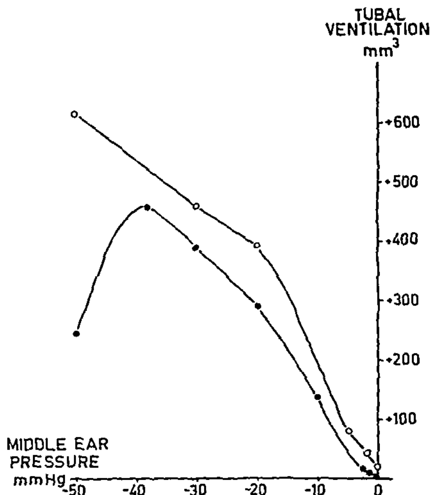


Fig 2
● — ● -aspiration
O — O +inflation (+1 mm Hg)

2 The "Locking" Mechanism

After direct application of negative pressure in the closed ear manometer system the tubal function during swallowing was studied, and every passage of air was recorded as a pressure decrease towards ambient pressure

Results and Comments

Normals

In normal subjects the 'locking' of the tube, making aspiration of air during deglutition impossible, could in some cases be provoked by a middle ear pressure of —30 mm Hg, while in others this phenomenon did not take

pressure than the corresponding volumes produced by "aspiration" only As judged from both these volume curves, the tubal ventilation during swallowing is more than three times greater after "aspiration" + inflation (+5 mm Hg) than after "aspiration" only within the middle ear pressure range from —20 to —30 mm Hg

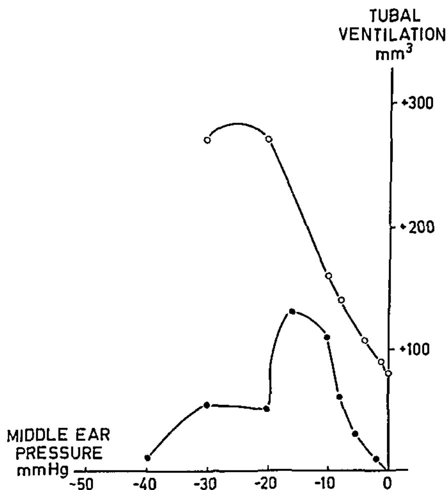


Fig 1
Tubal air passages by "aspiration and "aspiration + inflation
● — ● "aspiration
O — O " „ +inflation (+5 mm Hg)

In Fig 2 from another normal ear case, the pressure-volume curves show similar tendencies as in case one Here the inflation is produced by a positive pressure of +1 mm Hg during Toynbee

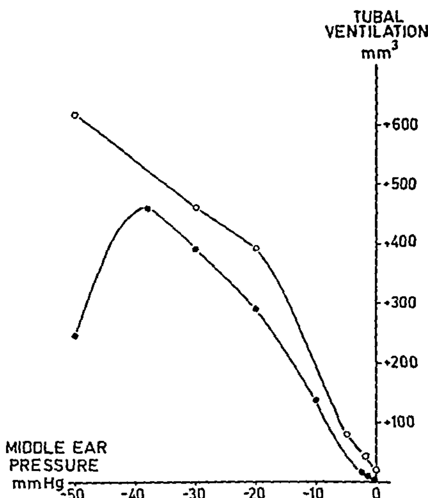


Fig 2
● — ● "aspiration"
O — O " „ "+inflation (+1 mm Hg)

2. The "Locking" Mechanism

After direct application of negative pressure in the closed ear manometer system the tubal function during swallowing was studied, and every passage of air was recorded as a pressure decrease towards ambient pressure.

Results and Comments

Normals

In normal subjects the "locking" of the tube, making aspiration of air during deglutition impossible, could in some cases be provoked by a middle ear pressure of —30 mm Hg, while in others this phenomenon did not take

place until at —50 mm Hg or below If now the subject tries to open the tube by swallowing no air enters the ear During the act of swallowing, a negative pressure change spike of short duration is sometimes recorded in the closed ear-manometer system, by which the original negative pressure in the system is momentarily accentuated during the attempted opening of the tubal lumen Thus for example, each spike represents a negative pressure change of about —0 2 mm Hg in the closed ear manometer space of 11 ml air volume at a middle ear pressure of —30 mm Hg

"Common cold" patients all showing normal ear drums at otoscopy

If a negative pressure is applied to the closed ear space according to the principles described above, the result will be a "locking" of the tube even when a small pressure has been applied in comparison with that needed for "locking" on normals Not seldom, this phenomenon is provoked even by a middle ear pressure from —5 to —10 mm Hg Further, in the "common cold' cases the negative pressure pulses are bigger, i e —1 to —2 mm Hg, and are more commonly recorded during deglutition than in the normal ear cases (see Fig 3) This phenomenon must be produced by a sudden increase

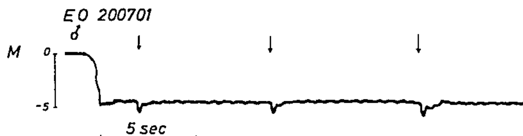


Fig 3
Negative pressure spikes produced by swallowing in a "common cold case during the "locking period
M=pressure change recordings in the closed ear space
↓ ="open nose deglutition

of the active ear space volume by different degrees of separation of the mucous membranes "glued together" at the aural end of the isthmus This separation is caused by the muscular opening forces acting on the tube The tube is then previously "locked" by a moderate intra aural negative pressure, probably owing to swelling of the mucous membranes of the tube, and the muscular opening forces are consequently only opposed by a small middle ear negative pressure In normal cases, "locking" is, however, obtained only by a pressure far below that of the atmosphere Thus the small negative pressure change spikes then obtained on normals may be explained by assuming that in these cases the greater negative pressure needed for "locking", should then more effectively oppose the muscular forces attempting to open the lumen of the isthmus

3. *Surface Tension Experiment*

The principle of von Neergaard's lung experiments has been applied on cases of chronic otitis with central drum perforations, provided that the tubal function has been found to be normal

Results and Comments

If an increasing positive pressure is applied directly in the ear cavity, it has been possible to force open the tube with air at an overpressure of about 20 mm Hg When corresponding experiments were carried out with sterile saline solution after elimination of the hydrostatic pressure, the tube could be forced open with the fluid already at a pressure of about +5 mm Hg

Thus it appears that surface tension takes part in the lumen opening mechanism of the Eustachian tube

Model

On the basis of the above results a simplified theoretical ear-Eustachian tube model has been sketched, with the aid of which some analogies will be drawn between the biological and the physical system

Principles

As appears from the sketch in Fig 4 the model consists of a "middle ear" chamber (ME) whose top opening is closed with a rubber membrane, i e the "tympanic membrane" (TM) At ambient pressure inside and outside the ME this occupies a volume of 1 ml If needed, a "rigid air cell system" (AC), volume 9 ml, can be airtightly connected to the ME-space The "tube" consists of a cone shaped semicircular rod, whose narrowest part is fastened to the ME, i e at the "isthmus" A thin rubber film is distended over the convex surface of the rod, while it is airtightly fastened to the back side The tension of the rubber film over the convexity is assumed equal along the whole of the rod at atmospheric pressure At the "isthmus" a small portion of the rubber film (ETM) leaves the rod and is airtightly fixed to that part of the circumference of the ME tube which is not connected to the rod An airtight rigid chamber (TCH) is mounted around the "tube" in such a way that "the rhinopharyngeal orifice of the tube" is open to the atmosphere at RH A main homogeneous layer of mucus is assumed to be applied between the rubber film and the convex surface of the rod Thus under normal pressure conditions, the lumen of the "tube" is closed The lumen will be opened by applying a negative pressure inside the TCH, which causes the rubber film to be sucked away from the rod surface Since mucus has been applied between the rod and the rubber film, surface forces acting at the liquid air interface may contribute to the elastic recoil of the rubber film Now it is well known that

the smaller radius of curvature, the greater the pressure opposing surface tension must be, i e. the amount of pressure needed for opening must increase in the aural direction of the "tube".

An attempt will now be made to reproduce, on this model, the results obtained from the patients In this way some biological phenomena may be explained

Experiment 1

Initial conditions ME+AC=10 ml, TM in the normal position, i e at ambient pressure If a negative pressure is applied in the TCH, the "tubal lumen" will be opened, beginning at RH, and with the aid of an increasing amount of negative pressure applied in the TCH it will be possible to establish air contact with the ME Before a complete tubal opening has been produced, the mobile rubber wall, ETM, will have been sucked in a little into the TCH by a negative pressure applied in this chamber The volume displacement of ETM would then increase the closed total volume of ME+AC, resulting in a negative pressure in this system if it were a rigid chamber In the circumstances, however, this negative pressure is easily compensated for by a slight inward movement of TM.

When bringing the negative pressure in the TCH back to ambient pressure the tubal lumen begins to close, starting at the "isthmus" If a mucus of higher surface tension is applied in the model it is evident that a greater amount of negative pressure must be applied in the TCH in order to make the tube open

Experiment 2

Initial conditions ME+AC=10 ml, pressure —10 mm Hg, sucking TM inwards, as on man, to a state of about 60 % of its maximum volume displacement capacity Further, the mobile ETM is sucked in a little into the ME-space, since the TCH is kept at ambient pressure

In order to make the tube open, a negative pressure in the TCH must in the first place be brought below the original negative pressure level of the ME+AC-system During this procedure, however, the ETM is simultaneously sucked out a little from the ME space, thereby producing a further lowering of the ME+AC-pressure During these experimental conditions, however, the resulting lowering of this pressure is very small owing to the fact that the increase of volume, caused by the ETM displacement, takes place within a closed ME+AC-space of great air volume Consequently, it should be comparatively easy to make the tube establish a free air contact between the ME and RH spaces

In connection with "common cold", "locking" of the tube has been demonstrated at moderate intra aural negative pressure levels Further, during deglutitions negative pressure change spikes have been recorded, representing an amount of pressure from —1 to —2 mm Hg Now if a pressure change of —1 mm Hg is produced in a 10 ml rigid chamber, the increase of the closed

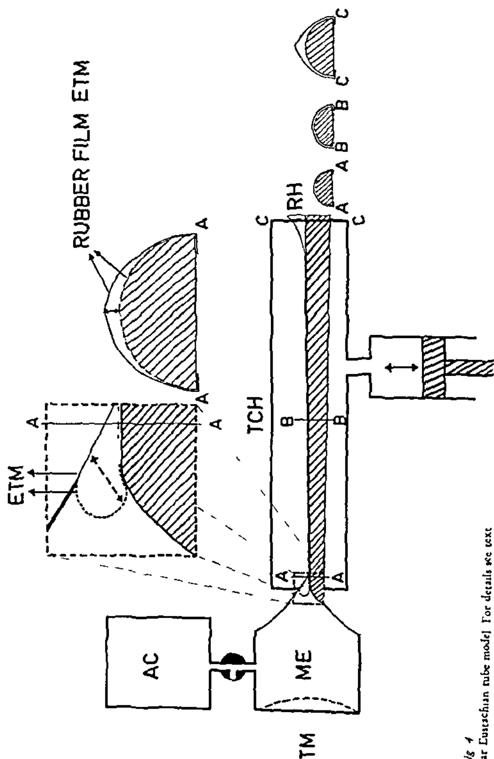


Fig 4
Ear Eustachian tube model For details see text

air space required for giving such a pressure change can be calculated, i e 13 mm³ Thus under these conditions it should still be possible to establish a free airway through the tube If the above mentioned attempted opening is combined with active inflation of air through the RH opening of the tube, and if the ability of both pressures to make middle ear ventilation are always compared at identical pressure differentials between the ME- and RH-spaces, it turns out that far smaller "opening forces" need to be applied around the "tube" in order to produce a more effective ventilation of the ME space by "aspiration" + inflation, as compared with "aspiration" only This is done even at small positive inflation pressures

Experiment 3

Initial conditions Only ME, 1 ml Otherwise exactly the same experimental conditions as in experiment 2

When trying to open the tube, a much lower negative pressure must be applied in the TCH Now if the ETM should be moved out of the ME-space, corresponding to a volume displacement of 13 mm³, this would increase the original ME pressure from —10 mm Hg to —20 mm Hg, supposing ME to be a rigid chamber In fact, within this pressure range, there is but a small chance left of a TM volume displacement by an inward movement, so the TM has only a slight possibility of reducing the negative pressure change spike occurring during the attempt to open the tube We are now faced with a vicious circle which can only be broken by active inflation of air through the RH opening of the tube

Experiment 4

Initial conditions ME+AC 10 ml, completely filled with water, TM in the "—10 mm Hg position"

When trying to open the tube under these conditions the opening forces, applied in the TCH, oppose themselves even more effectively than in experiment 3 and no air enters the ME+AC system, since all fluid is incompressible The only remaining way, except a drum puncture, is then active inflation through the RH opening of the tube Whether such a procedure succeeds or not is entirely dependent on the actual drum characteristics present, i e elasticity and mobility Similar ideas have been reported by Stevens in 1962, after experiments on an ear tube model filled with water Now if the model is supposed to contain thick viscous mucus some other factors may contribute to prevent mucus from escaping through the tube, and also air from entering the ear space, i.e the high viscosity and the high surface tension of the mucus

Discussion and Conclusions

Opinion differs widely as to whether, and to what extent, an impaired tubal function causes or maintains an inflammatory middle ear disease If we regard the serous otitis from this point of view, tubal occlusion was

originally regarded as the main cause of the disease This is the ex vacuo theory, according to Politzer (1862) and Bezold (1883) Contrary opinions have been put forward by several authors Thus Zöllner found that in most cases of serous otitis the tubal function is normal He arrived at this conclusion with the aid of his inflation method Further Zöllner quoted a number of investigators who have analysed middle ear effusions, indicating that the effusions are generally caused by inflammatory changes in the tympanum and are very seldom non inflammatory effusions, i e produced by vacuum Zöllner concludes that the serous otitis must be looked upon as a middle ear disease of an infectious or sometimes allergic origin involving all the mucous membranes, and that tube lesions only play a small part in serous otitis

The results of Senturia s experiments on dogs (1962) point in the same direction Making lesions on the nasopharyngeal tube orifices with electrocautery, Senturia regularly found that otitis followed these lesions, but no or only minimal inflammatory changes occurred in the lining membrane of the tube *indicating that "the Eustachian tube has a remarkable ability to recover* from trauma or infection"

Nevertheless, recent literature still contains information on "impaired tubal function" as "the key" of the inflammatory middle ear disease

In order to *elucidate these problems the following question would seem to* be of prime importance is the tubal middle ear ventilation during swallowing as effective if brought about by "aspiration" as if produced by inflation?

Some of the striking findings in this investigation are that at falling negative pressure in the ear, the normal ability of the tube to "aspire" is first more and more impaired, while inflation still takes place easily The reason for this must be that the "glued together" mucous membranes in the isthmus region are *sucked together even more tightly by the negative pressure present in the* middle ear On the other hand these membranes can be more easily separated by inflation This phenomenon is even more marked at "common cold", which *means that the tube is "locked" already by a very moderate intra-aural* negative pressure

Thus these investigations prove that the tubal lumen patency must not be regarded as accurately determined if studied by inflation only It seems very clear that the tube function must also be measured *with the "aspiration"* method This seems to be the natural way of elucidating the role played by the tube in the pathological states of the middle ear

Zusammenfassung

Bei genormten Ohr aspiration" experimenten an *gesunden Versuchspersonen* zeigt sich, dass die Luftmenge, die die Tube bei jedem Schluckakt *passiert,* schrittweise abnimmt auf Grund des Zunahme des *negativen* Druckes *im* Mittelohr Wenn gleichzeitig mit der "Aspiration" *die aktive Mittelohrbelüf*tung vorgenommen wird, ist die Luftmenge, welche *die* Tube *bei gleichem*

Druckunterschied zwischen Mittelohr und Nasenrachenraum passiert, wesentlich grosser als bei Versuchen, wobei nur die "Aspirationsmethode" angewandt wird

Bei "common cold" kann die Tube schon bei sehr niedrigem negativen Druck im Mittelohr verschlossen sein Wenn in diesem Zustand des Tubenverschlusses Versuche gemacht werden, die Tube durch den Schluckakt zu offnen, konnen gleichzeitig weitere negative Druckschwankungen innerhalb des abgeschlossenen Mittelohrraumes registriert werden, die durch Bewegungen hervorgerufen werden, welche durch den muskularen Offnungsmechanismus der Tube ausgelost werden Es tritt eine Aufhebung gegenseitiger Krafte ein Diese Phanomene werden an einem Mittelohr-Tubenmodell weiter erlautert

THE RELATIONSHIP OF MIDDLE EAR DISEASE TO MASTOID HYPOCELLULARITY

A working hypothesis

KNUT FLISBERG, SVEN INGELSTEDT and URBAN ORTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch, M.D), University of Lund

Human ears with poorly developed or undeveloped air cell systems are particularly susceptible to infection. Attempts have been made to define some physical factors related to the ear Eustachian tube system The incidence of inflammatory middle ear disease seems to be favoured by these factors under certain conditions, as in the hypocellular ear state.

It seems generally accepted that inflammatory middle ear diseases occur more commonly in the small air cell system than in the larger Starting from this fact the authors would like to point out some factors which, if combined with infection or allergy, may be supposed to be particularly injurious in cases of hypocellular mastoid state

The different well known hypothetical causes of the variation in the extent of pneumatization, however, have not been considered to be of decisive importance for the construction of our working hypothesis

The Main Factors

1) The mucosa of the air cells is normally less vascular than the mucosa of the middle ear Thus, regional quantitative differences in the mucosal blood flow may lead to a non uniform diffusion of O_2 from the ear gas into the blood

During operations on normal middle ear cases it is generally easy, with the aid of a microscope, to observe a quantitative difference in the blood supply of the mucosa The mucous membranes lining the promontorium and the ear drum seem to be much more abundantly vascularized than the thin mucosa lining the accessory air spaces Unfortunately, no direct comparative study seems to be available to judge from otologic literature In the textbook of anatomy by Rauber-Kopsch (1955), however, such a comparison is made, and it is pointed out that "the mucosa of the mastoid cells is poorer in blood vessels" Furthermore, speculating on the possible functions of the accessory spaces of the ear, Tumarkin (1957) suggests that "it is not for nothing that the mastoid endosteum is so thin and poorly vascularized", i e the accessory air spaces may have a "reservoir action" Starting from an equation on gas absorption in the ear space, Herberts (1948) discusses the quotient surface-volume He concludes that "in cases with larger pneumatic cell system the reduction of the pressure in the middle ear must be greater" Herberts pays no attention to different degrees of vascularization in different mucosal regions of the ear.

According to the present authors, however, the reduction of pressure is not mainly due to the volume of the ear space and the surface area of the mucosa, but to the degree of vascularization

As long as the capillary blood has a lower partial pressure of O_2 than that of the enclosed ear gas, a certain volume of O_2 leaves the closed space when the tube has been blocked Owing to its higher vascularity, the main portion of this gas volume may then diffuse into the blood across the mucosa of the middle ear The amount of negative pressure produced by O_2 diffusion may thus be supposed to be greater and more rapidly developed when the accessory air spaces functioning as an air chamber are lacking

2) During a "common cold" the tube may be easily "locked" and when even a moderate intra-aural negative pressure has arisen every passage of air through the tube during deglutition is made impossible The size of the functioning air volume in combination with the actual drum characteristics of elasticity and mobility are then factors which may determine whether the muscular forces required to overcome the tubal obstruction are sufficient or not

A transient "locking" of the tube frequently follows "common cold" and may naturally affect any ear case Sometimes, however, quite irrespective of whether the air cell system is large or even non-existent, the middle ear is affected and the progress of infection is then governed by the virulence of the invading organism and by lowered local or general body resistance But in the ears with a small or without any pneumatic system, there is some further form of local predisposition to infection, causing a tendency to chronicity

Now the otoscopic picture, frequently showing grave lesions of the drum, has been found to be in sharp contrast to the "normal tubal function" when determined by tympanic inflation Owing to this finding, the rôle played by the tube in the inflammatory states of the middle ear has sometimes been underestimated

In order to get a clearer understanding of the tube in ear disease its function should be investigated with the "ear aspiration" method During the basic research studies it is necessary first to select cases of head cold and upper respiratory infection before any obvious middle ear affection appears

If this is done, it appears that owing to hyperemia and slight swelling of the tube mucosa some form of tubal occlusion takes place Next, a negative pressure arises in the closed ear space by gas diffusion to the blood

As shown by the present authors in a previous paper, a further small volume increase occurs within the closed ear space during the "locking" period This volume increase is caused by active opening of the upper isthmus lumen which results in a further negative pressure drop in the closed ear space at the very moment of swallowing This further "locking" effect during these pressure drops is more pronounced the smaller the volume of the pneumatic system and the more reduced the ear drum capacity for making volume displacements This implies that the muscular opening activity opposes

itself, the normal ear drum regulates the middle ear pressure more often and more intensively in ear cases with a small pneumatic system Sooner or later the resulting load on the elastic fibres within membrana propria will gradually *destroy the elastic properties of the drum, making it flaccid and permanently "retracted" The result is a vicious circle*

As shown in an earlier paper, transudation may occur as a result of a vacuum within the ear and probably a mucosal congestion will be produced in the same way These disturbances may facilitate an inflammation within the tube, invading the middle ear cavity In this new situation the patient may have great difficulties in driving secretion out of the middle ear, especially if the cavity is filled with fluid Now the patient's capacity for forcing air into the ear is entirely dependent on the elastic qualities and the mobility of the drum actually present, since fluid is incompressible

In some of the cases of chronic otitis the central drum perforation should perhaps be looked upon as the last remaining possibility of the organism itself to bring about a "healing" of the middle ear process

Conclusions

After an acute occlusion of the tube at "common cold" a negative pressure may be supposed to be more rapidly developed and to become more pronounced in the small air cell system of the ear than in the big one, since the main part of the O_2 diffusing from the ear to the blood should in both cases pass through the mucosa lining the middle ear cavity owing to its higher degree of vascularity A cell system may thus be supposed to function as an air chamber In this latter case it is then less often necessary to employ the drum function as a regulator of the middle ear pressure at short time occlusions of the tube thanks to the large air volume of the ear space Further in this way there is less danger that the elastic elements of the drum are damaged Furthermore it is observed that in people with "common cold" the tube is easily "locked" by swelling of its mucous membranes Thus after a slight negative pressure has been established in the middle ear, it is impossible for the patient to "aspire" air into the ear via the tube during ordinary swallowing Air passage, on the other hand can easily be produced by inflation

During the "locking" period negative pressure change spikes can be recorded in the closed ear cavity during each act of swallowing, directly corresponding to the moments of attempted tubal opening This mechanism is particularly *dangerous in cases where the volume of the pneumatic ear system is small and where the elasticity and mobility of the drum have earlier been largely used up by the permanent vacuum in the ear or have been completely destroyed* This implies that the muscular opening *forces*, when acting on the tube, oppose themselves.

According to the present authors, however, the reduction of pressure is not mainly due to the volume of the ear space and the surface area of the mucosa, but to the degree of vascularization

As long as the capillary blood has a lower partial pressure of O_2 than that of the enclosed ear gas, a certain volume of O_2 leaves the closed space when the tube has been blocked Owing to its higher vascularity, the main portion of this gas volume may then diffuse into the blood across the mucosa of the middle ear The amount of negative pressure produced by O_2 diffusion may thus be supposed to be greater and more rapidly developed when the accessory air spaces functioning as an air chamber are lacking

2) During a "common cold" the tube may be easily "locked" and when even a moderate intra aural negative pressure has arisen every passage of air through the tube during deglutition is made impossible The size of the functioning air volume in combination with the actual drum characteristics of elasticity and mobility are then factors which may determine whether the muscular forces required to overcome the tubal obstruction are sufficient or not

A transient "locking" of the tube frequently follows "common cold" and may naturally affect any ear case Sometimes, however, quite irrespective of whether the air cell system is large or even non existent, the middle ear is *affected and the progress of infection is then governed by the virulence of* the invading organism and by lowered local or general body resistance But in the ears with a small or without any pneumatic system, there is some further form of local predisposition to infection, causing a tendency to chronicity

Now the otoscopic picture, frequently showing grave lesions of the drum, *has been found to be in sharp contrast to the "normal tubal function" when* determined by tympanic inflation Owing to this finding, the role played by the tube in the inflammatory states of the middle ear has sometimes been underestimated

In order to get a clearer understanding of the tube in ear disease its function *should be investigated with the "ear aspiration" method During the basic research studies it is necessary first to select cases of head cold and upper* respiratory infection before any obvious middle ear affection appears

If this is done, it appears that owing to hyperemia and slight swelling of the tube mucosa some form of tubal occlusion takes place Next, a negative *pressure arises in the closed ear space by gas diffusion to the blood*

As shown by the present authors in a previous paper, a further small volume increase occurs within the closed ear space during the "locking" period This volume increase is caused by active opening of the upper isthmus lumen which results in a further negative pressure drop in the closed ear space at the very moment of swallowing This further "locking" effect during these pressure drops is more pronounced the smaller the volume of the pneumatic system and the more reduced the ear drum capacity for making volume displacements This implies that the muscular opening activity opposes

EAR DRUM MOVEMENTS FOLLOWING STIMULATION OF THE MIDDLE EAR MUSCLES

HANS-ERIC HOLST, SVEN INGELSTEDT and URBAN ORTEGREN
Lund, Sweden

From the Department of Otolaryngology (Head Prof Hj Koch, M D), University of Lund

A method designed for recording small ear drum movements is described and used for recording the movements following stimulation of the middle ear muscle reflex. The results show that this reflex is a contraction of both muscles simultaneously in normal ears, and thus verify some earlier findings. The threshold level is raised as compared with the results obtained with other methods. This can be explained as another type contraction following stimulation with more intense stimuli.

During the last few decades the reflex contraction of the middle ear muscles has interested many authors The aims of their investigations have differed widely Thus some authors have studied the muscle reflex from a purely physiological point of view, others have been more interested in the importance of those muscles for controlling the sound transmission through the middle ear, while a third group of authors have used the middle ear muscle reflex for studies of the physical properties of the middle ear mechanism

The *following methods have been used for an investigation of the middle* ear muscle reflex in man direct inspection of the stapedial tendon or the ear drum movements, measurement of the change in the sound transmission through the middle ear following a contraction of the muscles, measurements of the change in the acoustic impedance of the ear drum following the muscle contraction, and finally, direct measurement of the ear drum movements following the stimulation of the middle ear muscles

The results of these investigations may be summarized as follows

1) It seems definitely clear that the middle ear muscles function as a kind of automatic volume control and are thus of great importance for the sound transmission in the ear (Sherrick and Mangabeira-Albernaz, 1961, Ward, Selters and Glorig, 1961, Ward, 1962)

2) It is possible to elicit a reflex contraction of the middle ear muscles by using different types of stimulus, such as tactile stimulation of the outer ear (Kato, 1913, Djupesland, 1961, Terkildsen, 1961), pain in the outer ear canal (Pichler and Bornschein, 1957, Klockhoff and Anderson, 1959), tactile stimulation of the orbital region (Klockhoff and Anderson, 1960), and acoustic stimulus (Luscher, 1929, Perlman, 1960, Metz, 1946, Møller, 1958, among others)

3) Metz and later Thomsen, 1955, state that the reflex threshold for an acoustic stimulus is proportional to the loudness of the stimulus sound In later investigations Møller, 1961, 1962, found that the reflex threshold is proportional to the sound pressure level of the stimulus sound

4) Jepsen, 1955, has shown that it is impossible to record any reflex in

cases of facial palsy where the injury also affects the stapedial nerve The authors refer to this fact when in later surveys they state that the function of the musculus tensor tympani in man is unknown (Thomsen, 1958, Klockhoff, 1961)

5) Metz, 1946, has shown that it is impossible to record any reflex contraction in cases of conductive hearing loss

6) According to Klockhoff *et al*, 1960, the muscle contraction following air blowing against the orbital region is a pure tensor contraction

7) Mendelson, 1957, and Terkildsen, 1960, have been able to demonstrate contractions in the musculus tensor tympani following acoustic stimulus

It is thus apparent from this summary that opinion is still divided as to the function of the musculus tensor tympani The reason for this is probably that it has not been possible, by the methods used, to distinguish a contraction in the musculus tensor tympani from a contraction in the musculus stapedius or these two from a contraction in both muscles simultaneously

If the sensitivity is high enough, the first mentioned method, direct inspection of the stapedial tendon or the ear drum movements (used by Kato, 1913, Luscher 1929, and later with direct inspection of the intact ear drum through a microscope or a lup, by Smith and Kawata), makes it possible to distinguish between a contraction of the musculus tensor tympani and a contraction of the musculus stapedius The change in sound transmission, however, cannot provide such information that it is possible to differentiate the two types of contractions Nor has such information been afforded by the change in the ear drum impedance A direct measurement of the ear drum movements following the muscle contraction, however, can provide information making possible a differentiation between the two muscles

Such direct measurements of the ear drum movements have been performed by Mangold, 1913, Kobrak, 1957, Mendelson, 1957, and Terkildsen, 1960 Mangold used a photomanometer giving the magnitude and direction of the ear drum movements, Kobrak small mirrors fastened on the ear drum measuring the movements as light beam deflections, Terkildsen a kind of fluid manometer with a droplet in a pipet as an indicator of the movements Mendelson used an electronic pressure transducer, measuring the pressure rise or drop caused by the ear drum movements The system is a closed one with a compensation for thermal influence By using the recording methods described here Mendelson and Terkildsen demonstrated a contraction of the musculus tensor tympani in man These results agree with those obtained by other authors in animal experiments

Present Investigations

The aim of this investigation was, first, to study ear drum movements following stimulation of the middle ear muscles, second, to examine these movements in order to search for a possibility to distinguish between tensor and stapedial contractions, and, finally, to compare these data obtained with

those obtained in earlier investigations These investigations were made possible by the work on the function of the Eustachian tube by Ingelstedt and Ortegren, which resulted in an equipment capable of measuring small movements of the ear drum

Methods

The magnitude and the direction of the ear drum movement is measured either as the rate of a gas flow caused by the ear drum movement through a system open to free air, or as the pressure change caused by the ear drum movement in a closed system

The flow rate meter device

If an air resistor is placed in a gas flow it causes a pressure rise on the upstream side and a pressure drop on the downstream side of the resistor The pressure rise and drop are proportional to the flow rate through the resistor The flow meter device used here operates on that principle It consists of an electronic pressure transducer connected to the ear with fine plastic tubes After amplifying the output voltage of the transducer is continually recorded on an impulse recorder

The pressure transducer is of a standard type, with a working pressure range of ±50 mm H_2O (EMT 573, Elema Schonander AB, Stockholm, Sweden) The impulse recorder is a two channel ECG recorder (Mingograph, Elema-Schonander AB, Stockholm, Sweden) This recorder can be used either with a DC-amplifier or with an AC-amplifier (See Fig 1)

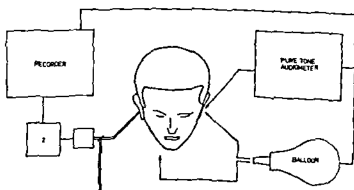


Fig 1
Block diagram of the experimental device
1 The electronic pressure transducer
2 Preamplifier

The pressure recording system

According to Boyle's law, the pressure rise or drop in a closed system is proportional to the volume change if the temperature is kept constant.

The pressure recording system consists of the same transducer as that used in the gas flow rate meter device It is connected to the ear in the same way and the only real difference in the two systems is that the passage through the resistor is closed in the pressure recording system

The response

When the flow rate meter device is used, the response shows the magnitude and the direction of the ear drum movement, but not the position after the movement has stopped, curves B and D in Fig 2. When the pressure recording

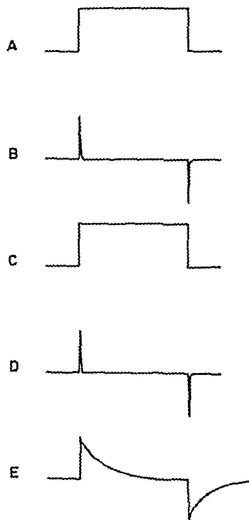


Fig 2
The shape of the recordings using the various recording principles.
A Square wave pressure impulse to be recorded
B With flow rate meter device and DC amplifier
C With pressure recording device and DC amplifier
D With flow rate meter device and AC-amplifier
E With pressure recording device and AC amplifier

system is used, the response shows the magnitude and the direction and also the position of the ear drum after the movement has stopped, curves C and E in Fig 2 From the Fig it is also seen how the choice of amplification modifies the responses, B and C, DC-amplifier and D and E, AC-amplifier

Sensitivity

The sensitivity of the recording device is as follows with the maximal available DC-amplification the gas flow produced by a pressure of 6 mm H_2O gives a 30 mm deflection, with the maximal available AC-amplification this deflection is obtained when the gas flow is produced by only 2 mm H_2O These values refer to recordings with the gas flow meter device Using the closed manometer system, a pressure of 0 5 mm H_2O gives a deflection of 40 mm The frequency response is linear up to 20 Hz at DC amplification Above this frequency the response decreases in amplitude and is 75 per cent of maximal amplitude at 40 Hz.

Stimuli

The stimuli used to elicit the reflex contraction are of three types tactile stimulation of the orbital region by air blowing ad modum Klockhoff, tactile and/or acoustic stimulation of the contralateral ear by air blowing ad modum Djupesland, acoustic stimulation of the contralateral ear with pure tones, in all cases with 500 Hz 127 dB above 0 0002 dyne/sqcm, in some cases also with other frequencies

Equipment

The air blowing was performed with a rubber balloon The stimulus strength obtained was high Air blowing against the ear gives a stimulation with a sound very similar to a white noise (Djupesland) This type of stimulus should therefore be regarded as an acoustic stimulation

The acoustic stimulation with pure tones was performed by means of a pure tone audiometer with air conducted sound The audiometer used was an Amplivox model 61, specially calibrated so as to give higher intensities than usual

All types of stimuli were recorded on one channel of the recorder The amplification is not standardized

Performance

The outer ear canal is airtightly sealed with a rubber cuff around the end of the tube and the cuff is lodged in the bony part. As resistor a plastic tube is used, 300 mm long and with an inner diameter of 1 mm, and connecting the pressure transducer with the free air The subject was placed in an ordinary examination chair with his head firmly fixed. This arrangement made it possible to avoid disturbances from involuntary movements by the subject

Material

The investigation was performed on 16 normal subjects, all with normal ears and normal hearing, besides, patients with various types of injuries were examined Most of them had chronic otitis, one otosclerosis, and two cases had a broken ossicular chain later verified at operation Finally, there were cases with facial palsy of a peripheral type

Results

Interest was here focussed on the normal series The responses were of three types (Fig 3).

1) Outward movement of the ear drum,
2) Inward movement of the ear drum,
3) Outward-inward biphasic movement or an inward-outward biphasic one, *here and in the following simply called biphasic response*

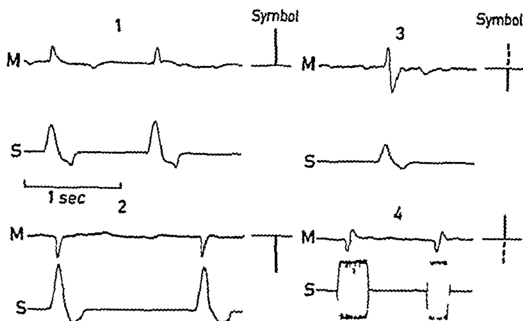


Fig 3
The recorded drum movements.
M = meatal recording S = stimulus recording

= initial direction at biphasic movement.

1 Outward movement.
2 Inward movement.
3 Outward inward biphasic movement.
4 Inward-outward biphasic movement.

The results in the normal series are brought together in Fig 4 The lines here only represent the direction of the movement, not the actual magnitude From the Fig. it is thus possible to compare the responses for each subject to the three types of stimulus used

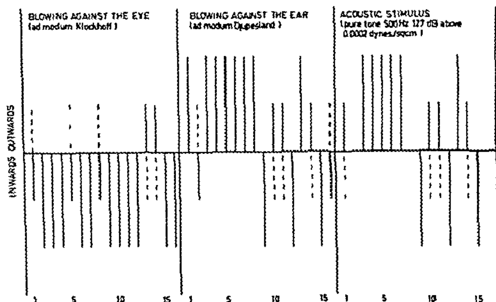


Fig 4.
The recorded responses in the normal series. Only the direction of the movements is indicated. The numbers refer to the subjects. The symbols are decoded in Fig 3

= initial direction at biphasic movement

It is seen that in most cases blowing against the eye gives an inward movement of the ear drum Biphasic responses are, however, comparatively common The movements following blowing against the ear are mainly outward movements, but here also the other types of responses are common The acoustic stimulus with pure tone gives responses with about the same distribution as blowing ad modum Djupesland

The reproducibility of the responses, tested with repeated stimuli, was of a very high degree, identical responses, both in type and magnitude, could be reproduced any number of times. The reproducibility for repeated examinations at varying intervals was also of a high degree Both ears of one of the subjects were examined and showed the same type of response

The latency time, defined as the time delay from the onset of the stimulus to the onset of the response, is slightly longer *for a stimulation of* the eye than for the other types of stimulus, see Fig 5. In the Fig the latency times given in earlier investigations are also indicated

ments of the ear drum In some of the cases it was possible to record a reflex contraction with a modified acoustic impedance measuring method The threshold level was then 15—20 dB higher than normal in the healthy contralateral ear

Discussion

The types of response agree with those observed by Mendelson A close comparison, however, is impossible since Mendelson has not published any curves

All the stimuli used in this work are of such intensity that they give *ward off or startle reactions The middle ear muscle reflex itself is probably* such a startle reaction The more widespread reactions in the face and neck muscles can interfere with the recordings of the very small ear drum movements The following is proof that the responses are pure ear drum movements and not startle reactions

1) The qualities of the responses agree with earlier results

2) It is possible to reproduce the responses at repeated examinations

3) The results from injured ears show a logical relation to anatomical and pathological data

To sum up the results from normal series, it is obvious that the various types of stimuli give responses typical of each particular kind of stimulus In *about one third of the subjects, however, biphasic responses are obtained Now* if the ear drum movement following the stimulation of the middle ear muscles is caused by the muscle contraction, it is clear that these biphasic responses are contractions of both muscles In the case of the contraction following air blowing against the ear and acoustic stimulation, the responses show a distribution without a sharp maximum These results justify the conclusion that the contraction following the stimuli used here is a contraction of both muscles in normal ears Also from a physiological point of view this is logical and agrees well with the results obtained in experimental work on animals (Wiggers 1937 Wersall, 1958) and verifies the results of experiments on man by Mangold Terkildsen, and Mendelson

The threshold level for acoustic stimulation with pure tones is slightly raised as compared with values obtained with a modified acoustic impedance measuring method This can be explained by assuming a lower sensitivity for the method described here. It is also possible however, that there is a difference in the contraction itself For moderate stimulus levels the contraction is a pure isometric one without any displacement of the ossicles and the ear drum, resulting only in a change of the acoustic impedance and transmission, while at a more intense stimulation the stronger muscle dominates, which results in displacement of the ossicles and the ear drum, giving the responses recorded here

The disappearance of the inward component of a biphasic response following *stimulation with quickly repeated tone bursts can be explained as fatigue of*

the tensor muscle From earlier investigations it is known that the tensor muscle has a longer latency time than the stapedial muscle, which can also explain the phenomenon observed here

It is shown that in cases with only the tensor muscle working the threshold is higher than in normal cases It seems to be the first time that an isolated contraction of the tensor muscle is observed in man

Conclusion

The results thus seem to admit of the following conclusions

1) It is possible to record the ear drum movements caused by the middle ear muscle reflex

2) The middle ear muscle reflex is a contraction of both muscles simultaneously

3) The threshold for eliciting a contraction of the tensor muscle is 15—20 dB higher than the normal threshold

4) After standardization of the method, it can be of clinical value in diagnosis of conductive hearing loss

Zusammenfassung

Es wird eine Methode beschrieben fur die Feststellung kleiner Trommelfellbewegungen und deren Anwendung fur die Registrierung der Bewegungen nach der Auslosung des Reflexes der Mittelohrmuskeln. Die Resultate zeigen, dass dieser Reflex im Normalfall eine gleichzeitige Kontraktion beider Muskel auslost, was die Ergebnisse einiger fruheren Untersuchungen bestatigt Die Ergebnisse nach Anwendung dieser Methode werden mit den Resultaten anderer Methoden verglichen und die benotigten Schwellenwerte liegen hoher als bei fruheren Methoden Unterschiede werden durch eine andere Art der Muskelkontraktion erklart, die auf starkere Reize erfolgen kann

Acknowledgements

The authors' thanks are due to many people who have kindly facilitated these studies

Mr R ELMQVIST, M D , Elema Schonander AB, Stockholm, for valuable discussions and technical advice

Mr S G OLSSON and Mr B ANDERSSON, Engineers at Elema Schonander AB, Malmo, for cooperation and technical assistance

Mr G CRONA, Engineer at the Hospital of Lund, for manufacturing different technical devices

Mrs L ORTEGREN for drawing the illustrations

Mr J NILSSON, Senior Photographer, for making illustrations

All members of the ENT Department for valuable help

These investigations were supported by grants from the Swedish Medical Research Council, the Medical Faculty, University of Lund, and the Maggie Stephen Foundation, Sweden

Lund, March, 1963

Knut Flisberg
Hans Eric Holst
Sven Ingelstedt
Urban Ortegren

References

ADAMS, W S, 1953 Clinical otomanometer *J Laryng*, *67*, 488

ANDERSON, H., HOLMGREN, L, and HOLST, H.-E., 1956 Experiments with an objective method of testing the middle ear function. *Acta oto-laryng*, *46*, 38

— 1956 Tympanometri. Presented at the Annual Meeting of the Swedish Otologic Society

ARMSTRONG, H G, and HEIM, J W., 1937 The effect of flight on the middle ear *J Amer med Ass*, *109*, 417

ASCHAN, G., 1948 Aero-otitis media and aerosinusitis. Experimental histo-pathological changes due to oxygen deficiency and oxygen poisoning *Acta oto-laryng*, *Suppl* 69, 1

— 1954 The Eustachian tube *Acta oto-laryng*, *44*, 295

— 1955 The anatomy of the Eustachian tube with regard to its function. *Acta Soc Med Upsalien*, *60*, 131

BARBEY, K OLTERSDORF, U, and PAUSCHINGER, P, 1958 Eine Elektrische Registriermethode fur die Beurteilung der Durchgangigkeit der Tuba Eustachii. *Dtsch med Wschr*, *83*, 39

BEHNKE, A R., 1945 Physiologic effect of pressure changes with reference to otolaryngology *Arch Otolaryng*, *42*, 110

BEZOLD, F, 1883 Die Verschliessung der Tuba Eustachii. *Berl klin Wschr*, *20*, 551

CANFIELD, N, and BATEMAN, G H 1944 Myringopuncture for reduced intratympanic pressure Report of pressure chamber experiments *J Aviat Med*, *15* 340

CHANG, H T, MARGARIA, R, and GELFAN S, 1950 Pressure changes and barotrauma resulting from decompression and recompression in the middle ear of monkeys. *Arch Otolaryng*, *51*, 378

CLELAND 1869 On the question whether the Eustachian tube is opened or closed in swallowing *Journal of Anatomy and Physiology* *3*, 97

COMPERE W E. 1958 Tympanic cavity clearance studies *Trans Amer Acad Ophtal Otolaryng* *62* 444

DIAMANT M 1940 Otitis and pneumatisation of the mastoid bone *Acta oto-laryng*, *Suppl* *41*, 1

— 1962 Mastoid pneumatisation and its function. *Arch Otolaryng* *76*, 390

v DISHOECK H A E. 1937 Das Pneumophon. *Arch Ohr*, *Nas u Kehlk Heilk*, *144*, 55

— 1939 Theorie und Praxis des Pneumophons. *Arch Ohr Nas u Kehlk.-Heilk.*, *146* 5

— 1941 Negative pressure and loss of hearing in tubal catarrh *Acta oto laryng* *29*, 303

— 1944 Stenosis of the Eustachian tube *Acta oto-laryng* *32*, 346

— 1947 Resistance measuring of the Eustachian tube and the ostium and [illegible] valve mechanism *Acta oto laryng* *35* 317

— 1948 Hydrotympanon in the different clinical aspects of the tubo-tympanical catarrh. *Acta oto-laryng*, *36*, 429

— 1952 Tubal disorders and the pneumophone *Acta oto laryng*, *41*, 196

DJUPESLAND G, 1961 Registrierung von intra-auralen Muskel reflexen durch [illegible] Reizung bei Menschen *Acta oto-laryng* *53* 397

— 1962 Intra aural muscular reflexes elicited by air current [illegible] of the external ear *Acta oto-laryng* *54* 143

FLODGREN E., 1943 En apparatur for mätning av trumhåletrycket. *Nord Med.*, *[illegible]*, 2239

FOWLER, E. P, 1920 Drum tension and middle ear air pressures, their determination, significance and effect upon the hearing *Ann Otol*, *29*, 688

FOWLER, E P Jr, 1945 Causes of deafness in flyers *Arch Otolaryng*, *42*, 21

FRENCKNER, P, 1939 Movements of the tympanic membrane and of the malleus in normal cases and in cases of otosclerosis *Acta oto laryng*, *27*, 587

GRAVES, G O, and EDWARD, L E, 1944 The Eustachian tube *Arch Otolaryng*, *39*, 359

v GYERGYAY, A, 1932 Neue Wege zur Erkennung der Physiologie und Pathologie der Ohrtrompete *Mschr Ohrenheilk.*, *66*, 769

HANDL, K, 1954 Direkte Messungen des Luftdruckes der Mittelohrraume bei Meerschweinchen *Arch Ohr -, Nas , u Kehlk. Heilk.*, *165*, 408

— 1957 Der Einfluss der Musculus tensor tympani und der Ohrtrompete auf den Mittelohrluftdruck beim Meerschweinchen *Arch Ohr , Nas , u Kehlk.-Heilk.*, *170*, 473

HARTMANN, A, 1879 Experimentelle Studien uber die Funktion der Eustachischen Rohre Veit Co Leipzig

HERBERTS, G, 1948 A study of the function of tuba Eustachii *Upsala lakareforenings forhandlingar* *53*, 293

HOLBOROW, C A, 1962 Deafness associated with cleft palate *J Laryng*, *76*, 762

HOLMGREN, G, 1934 Recherches experimentales sur les fonction de la trompe d Eustache Communication prealable *Acta oto-laryng*, *20*, 381

HOLMGREN, L., 1940 Experimental tubar occlusion *Acta oto laryng*, *28*, 587

— 1941 Tubar occlusion, an answer to N Rh. Blegvad *Acta oto laryng*, *29*, 313

JEPSEN, O, 1955 Acoustic stapedius reflex in man. Universitetsforlag, Aarhus

JUGANOV, E. M, 1960 Orontrumpetens fysiologi *Vestn Oto rino laring*, *22*, 34

KATO, T, 1913 Zur Physiologie der Binnenmuskeln des Ohres. *Plugers Arch ges Physiol*, *150*, 569, (cited by Pichler, H, and Bornschein H)

KAWATA, S., 1958 Judgement of recruitment by means of measuring the retraction grade of the tympanic membrane *Acta oto laryng*, *49*, 517

KLOCKHOFF, I., 1959 Bekesy audiometry and recruitment of loudness. Presented at the Annual Meeting of the Swedish Otologic Society

— 1961 Middle ear muscle reflexes in man. *Acta oto laryng*, Suppl 164

KLOCKHOFF, I., and ANDERSON, H, 1959 a Registrierung des cutanen Stapediusreflexes durch akustische Impedanzmessung *Arch Ohr , Nas -, u Kehlk. Heilk.*, *175*, Heft II, 484

— 1959 b Recording of the stapedius reflex elicited by cutaneous stimulation. *Acta oto laryng*, *50*, 451

— 1960 Reflex activity in the tensor tympani muscle recorded in man *Acta oto-laryng*, *51*, 184

KOBRAK, H., 1957 Objective audiometry *Arch Otolaryng*, *65*, 26

KOTTMEYER, G, 1958—59 Eine Einfache Methode zur Prufung der Tubendurchgängigkeit *Z. Hals , Nas. u Ohrenheilk.*, *7*, 149

KURATA T 1937 Rontgenologische Untersuchungen uber die pneumatischen Zellen des Schlafenbeines. *J Med Assoc Formosa*, *36*, (cited by Diamant M., 1940)

LIEBERMANN, A. T., 1946 Aero-otitis media in pressure chamber 'flights *Arch Otolaryng*, *43*, 500

LINK, R., and HANDL, K., 1955 Die Pneumatisation des Schlafenbeines ein Schutz gegen Larmschwerhorigkeit. *Arch Ohr , Nas , u Kehlk. Heilk.*, *167*, 610

LOEB, M, and FLETCHER, J L., 1961 Contralateral threshold shift and reduction in temporary threshold shift as indices of acoustic reflex action. *J acoust Soc Amer*, *33*, 1558

LOCH, W E., 1942 Effect of experimentally altered air pressure in the middle ear on hearing acuity in man. *Ann Otol*, *51*, 995

LUCAE, A. 1864 Ueber die Respirationsbewegungen des Trommelfells. *Arch Ohrenheilk.*, 1, 96

— 1867 Zur Function der Tuba Eustachii. *Arch Ohrenheilk.*, *3*, 174

— 1875 Zur Function der Tuba Eustachii und des Gaumensegels. *Archiv fur pathologische Anatomie*, 64, 476.

LUSCHER, E., 1929, 1929/30 Die Funktion des Musculus stapedius beim Menschen I, II. Z. Hals, Nas- u. *Ohrenheilk.*, 23 (1929), 101, 25 (1930), 462

MACBETH, R, 1960 Some thoughts on the Eustachian tube *Proc roy Soc Med*, 53, 151

MCGIBBON, J E G, 1942 Aviation pressure deafness. *J laryng*, 57, 14

— 1942 The nature of the valvular action (passive opening) of the Eustachian tube in relation to changes of atmospheric pressure and to aviation pressure deafness. *J laryng*, 57, 344

MCMYN, J K, 1940 The anatomy of the salpingo-pharyngeus muscle. *J laryng*, 55, 1

MACH, E., and KESSEL, J, 1872 Die Function der Trommelhohle und der Tuba Eustachii. *S B Akad Wiss Wien math nat Kl*, 3, 329

— 1872 Versuche über die Accommodation des Ohres. *S. B Akad Wiss Wien math-nat Kl*, 3, 337

MAGNUS, A, 1864 Beobachtungen uber das Verhalten des Gehör Organs in komprimierter Luft. *Arch Ohrenheilk.*, 1, 269

MANGOLD, E., 1926 Das äussere und mittlere Ohr und ihre physiologischen Funktionen Handb der Norm und Pathol Phys.

MEAD, J, WHITTENBERGER, L., and RADFORD E. P, JR, 1957 Surface tension as a factor in pulmonary volume — pressure hysteresis *J appl Physiol*, 10, 191

MELLVILL JONES G 1961 Pressure changes in the middle ear after altering the composition of contained gas. *Acta oto laryng* 53 1

MENDELSON, E S, 1957 A sensitive method for registration of human intratympanic muscle reflex. *J appl Physiol*, 11, 499

— 1961 Improved method for studying tympanic reflexes in man *J acoust Soc Amer* 33, 146

METZ, O, 1946 The acoustic impedance measured on normal and pathological ears. *Acta oto laryng Suppl* 63 3

— 1951 Studies on the contraction of the tympanic muscles as indicated by changes in the impedance of the ear *Acta oto laryng*, 39, 397

— 1952 Threshold of reflex contractions of muscles of middle ear and recruitment of loudness *Arch Otolaryng* 55, 536

— 1953 Influence of the patulous Eustachian tube on the acoustic impedance of the ear *Acta oto laryng Suppl* 109, 105

MILLER, J B 1961 Patulous Eustachian tube *Arch Otolaryng*, 73, 310

MØLLER A R 1958 Intra aural muscle contraction in man examined by measuring acoustic impedance of the ear *Laryngoscope (St Louis)*, 68, 148

— 1960 Improved technique for detailed measurements of the middle ear impedance. *J acoust Soc Amer* 32, 250

— 1961 Network model of the middle ear *J acoust Soc Amer*, 33, 168

— 1961 Bilateral contraction of the tympanic muscles in man *Ann Otol*, 70, 735

— 1961 1962 Studies of speech and hearing Speech transmission Laboratory, Royal Instit. of Technology, Stockholm, Sweden, Quarterly Prog and Status Report, 4 (1961), 1 (1962)

— 1962 The sensitivity of contraction of the tympanic muscles in man *Ann Otol*, 71 86

NEERGAARD K, 1929: Neue Auffassungen uber einen Grundbegriff der Atemmechanik. *Z ges exp Med* 66 373

OLTERSDORF, U 1954 Eine Registrierungsmethode fur die Beurteiligung der Weite der Tuba Eustachii. *Arch Ohr-, Nas., u Kehlk.-Heilk*, 165, 399

— 1962 Die Elektrische Registrierung der Tubendurchgängigkeit. *Z. Laryng Rhinol*, 41, 161

PERLMAN, H B, 1939 The Eustachian tube *Arch Otolaryng*, 30, 212
— 1943 Quantitative tubal function *Arch Otolaryng*, 38, 453
— 1951 Observations on the Eustachian tube *Arch Otolaryng*, 53, 370
— 1960 Physiological basis for tubal function tests *Arch Otolaryng*, 71, 384.
— 1960 The place of the middle ear muscle reflex in auditory research *Arch Otolaryng*, 72, 201
PICHLER, H, and BORNSCHEIN, H, 1957 Audiometrischer Nachweis nichtakustisch ausgeloster Reflexkontraktionen der Intraauralmuskulatur *Acta oto laryng*, 48, 498
POHLMAN, A G, and KRANTZ, F W, 1923 The effect of pressure changes in the external auditory canal on acuity of hearing *Ann Otol*, 32, 545
POLITZER, A, 1861 Ueber eine Beziehung des Trigeminus zur Eustachischen Ohrtrompete *Phys Med Gesellschaft, Wurzburg*, 2, 92
RAUBER and KOPSCH, 1955 Lehrbuch und Atlas der Anatomie des Menschen Bd II Thieme Verlag, Stuttgart
ROGERS, R L, KIRCHNER, R F, and PROUD, G O, 1962 The evaluation of Eustachian tubal function by fluorescent dye studies *Laryngoscope (St Louis)*, 72, 456
SCHWARTZE, H, 1864 Respiratorische Bewegung des Trommelfelles *Arch Ohrenheilk.*, 1, 139
SENTURIA, B H, CARR, C D, and AHLVIN, R, 1962 Middle ear effusion pathologic changes of the mucoperiosteum in the experimental animal *Ann Otol*, 71, 632
SENTURIA, B H, GESSERT, C. F, CARR, C. D, and BAUMAN, E S, 1958 Studies concerned with tubotympanitis *Ann Otol*, 67, 440
SHERRICK, C E, and MANGABEIRA ALBERNAZ, P L, 1961 Auditory threshold shifts produced by simultaneously pulsed contra lateral stimuli *J acoust Soc Amer*, 33, 1381
SIEBENMANN, F, 1892 Horprufungsresultate bei reinem Tubencatarrh *Z Ohrenheilk*, 22, 308
SILBIGER, H, 1950 Ueber das Ausmass des Mastoidpneumatisation beim Menschen *Acta anat (Basel)*, 11, 215
SMITH, H D, 1943 Audiometric effects of voluntary contraction of the tensor tympani muscles. *Arch Otolaryng*, 38, 369
STERNSTEIN, H J, and McGREGOR, J B, 1944 The effect of nasal ventilation upon tubal equalizing efficiency in flying personnel *J Aviat Med*, 15, 244
STEWART, C. B, WARWICK, O H, and BATEMAN, G L., 1945 Acute otitic barotrauma resulting from low pressure chamber tests *J Aviat Med*, 16, 385
STEVENS, D M, 1962 Long term effects of serous otitis *Lancet 7249*, 216
STREIT, H, 1922 Zur Diagnose der Verengung der Tuba Eustachii *Arch Ohr, Nas-, u Kehlk. Heilk.*, 110, 54
TERKILDSEN, K, 1960 An evaluation of perceptive hearing losses in children based on recruitment determinations *Acta oto laryng*, 51, 476
— 1960 Acoustic reflexes of the human musculus tensor tympani *Acta oto laryng, Suppl*, 158, 230
— 1960 The intra aural muscle reflexes in normal persons and in workers exposed to intense industrial noise *Acta oto laryng*, 52, 384
— 1961 Conduction of sound in the human middle ear *Arch Otolaryng*, 73, 69
— 1961 Taktile intra aurale muskelreflekser Speech for the Danish Otolaryng Society, May 1961
TERKILDSEN, K, and SCOTT NIELSEN, S, 1960 An electroacoustic impedance measuring bridge for clinical use *Arch Otolaryng*, 72, 339
TERKILDSEN, K, and THOMSEN, K A, 1959 The influence of pressure variations on the impedance of the human ear drum *J Laryng*, 73, 409
THOMSEN, K. A, 1955 Employment of impedance measurements in otologic and otoneurologic diagnostics *Acta oto laryng*, 45, 159
— 1955 Eustachian tube function tested by employment of impedance measuring *Acta oto-laryng*, 45, 252

— 1957 Studies on the function of the Eustachian tube in a series of normal individuals. *Acta oto laryng*, *48*, 516

— 1958 Akustisk Impedansmåling ved Funktionsundersøgelser af Tuba Eustachii og til Bestemmelse af Recruitment. Christtreus, Copenhagen.

— 1958 Investigations on Toynbee's experiment in normal individuals. *Acta oto-laryng*, *140*, 263

— 1958 Investigations on the tubal function and measurement of the middle ear pressure in pressure chamber *Acta oto laryng*, *Suppl 140*, 269

THULLEN, A, 1962 Die elektrische Registrierung der Tubendurchgängigkeit. *Z. Laryng Rhinol*, *41*, 169

TOYNBEE, J, 1853 On the muscles that open the Eustachian tube *Proc Roy Soc Med*, *6*, 286

— 1860 The diseases of the ear Churchill, London

TUMARKIN, A, 1957 On the nature and vicissitudes of the accessory air spaces of the middle ear *J Laryng*, *71*, 65, 137, 211

WARD, W D, 1962 Studies on the aural reflex. II. Reduction of temporary threshold shift from intermittent noise by reflex activity, implications for damage risk criteria. *J acoust Soc Amer*, *34*, 234

WARD, W D, SELTERS, W, and GLORIG, A, 1961 Exploratory studies on temporary threshold shift from impulses *J acoust Soc Amer*, *33*, 781

WERSALL, R., 1958 The tympanic muscles and their reflexes *Acta oto-laryng*, *Suppl 139*

WEVER E. G, BRAY, C W, and LAWRENCE M, 1942 The effects of pressure in the middle ear *J exp Psychol*, *30*, 40

WEVER, E G, and LAWRENCE, M, 1954 Physiological Acoustics Princeton, London

WEVER, E G, LAWRENCE, M, and SMITH, K. R, 1948 The effect of negative air pressure in the middle ear *Ann Otol* *57*, 418

WEVER, E. G, and VERNON, J A, 1956 The control of sound transmission by the middle ear muscles *Ann Otol*, *65*, 5

WIGGERS, H C., 1937 The function of the intra-aural muscles *Amer J Physiol* *120*, 771

WITTMAACK, K, 1918 Ueber die normale und die pathologische Pneumatisation des Schlafenbeines. Verlag G Fischer, Jena

WOYATSCHEK, W, 1908 Ein neuer hermetischer Ohrenobturator *Arch Ohr*, *Nas u Kehlk. Heilk*, *75—76*, 217

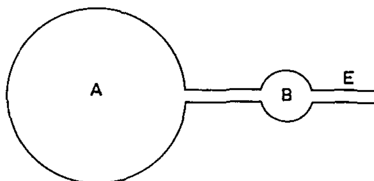
ZÖLLNER, F 1942 Anatomie, Physiologie, Pathologie und Klinik der Ohrtrompete Springer Verlag, Berlin

APPENDIX

Deduction and Discussion of the Volumetric Equation

GÖRAN LEIDE, Fil Dr,
Lund Sweden

For evaluating the accuracy of the methods used, the case on page 26 is illustrated by a model



A is a rigid tank, B the "middle ear" space and E the "Eustachian tube" through which the volume of gas quantitatively determined enters or leaves the system A+B The air inside A is kept at room temperature and at the same pressure and relative humidity as those of the ambient air, i e 760 mm Hg and about 50 % The air inside B is kept at body temperature and the relative humidity is 100 % Now if A and B are connected, the moist air inside B is after a long time mixed with the air inside A, thereby raising the relative humidity within A to 100 % At this point the air pressure in A+B would rise from 760 mm Hg to about 770 mm Hg During the experiment on patients, however, this factor must be negligible since the tube between A and B is fairly long and narrow Furthermore, in the course of experiments the ranges of temperature and pressure inside the system are small, and evaporation or condensation of moisture will have a negligible influence on the pressures recorded The ideal gas law is then applicable

The following symbols are used

V_a = volume of A space

V_b = volume of B space

v = volume of the air insufflated through E at the pressure P and the temperature T_b

P = pressure of the system A+B before insufflation

p = pressure increase in A+B

m_a = weight of air in A before insufflation

m_b = *weight of air in B before insufflation*

m = weight of air insufflated through E

R = ideal gas constant

M = *mean molecular weight of air*

T_a = absolute temperature inside A

T_b = absolute temperature inside B

The ideal gas law may be written $PV = \frac{m}{M} R\,T$

Solving for m and introducing the symbols above the equations for the weight of air in A and B become

$$m_a = \frac{M}{R} P \frac{V_a}{T_a} \tag{1a}$$

$$m_b = \frac{M}{R} P \frac{V_b}{T_b} \tag{1b}$$

The total weight is then obtained by

$$m_a + m_b = \frac{M}{R} P \left(\frac{V_a}{T_a} + \frac{V_b}{T_b}\right) \tag{1}$$

By insufflation an additional amount of air is introduced into the system but the volume does not change Most of this air goes into A and almost immediately gets the temperature T_a As this amount of gas is small compared with the total amount present in A, the temperature in A is still T_a or almost T_a Hence the expression $\frac{V_a}{T_a} + \frac{V_b}{T_b}$ is unchanged and the relation between mass and pressure becomes

$$m_a + m_b + m = \frac{M}{R} (P + p) \left(\frac{V_a}{T_a} + \frac{V_b}{T_b}\right)$$

Hence the amount of air introduced in A+B is expressed by

$$m = \frac{M}{R} p \left(\frac{V_a}{T_a} + \frac{V_b}{T_b}\right) \tag{2}$$

According to the general gas law, assuming pressure P and temperature T_b, the volume of this amount of air is

$$v = \frac{m}{M} \quad \frac{RT_b}{P} = \frac{p}{P} \left(\frac{T_b}{T_a} V_a + V_b\right) \tag{3}$$

The value of $\frac{M}{R}$ for air is 2 17 · 10³ $\frac{cm^3 \cdot torr}{gm \cdot °K}$ With volumes in cm³, *pressures in torr (mm Hg) and temperatures in °K, the mass m in (2) is* obtained in grammes and the volume v in (3) in cm³

The maximum error expected in v is due to the errors in p, P and V ($V = \frac{T_b}{T_a} V_a + V_b$) according to the formula $dv = \frac{\delta v}{\delta p} dp + \frac{\delta v}{\delta P} dP + \frac{\delta v}{\delta V} dV$

The partial derivatives are calculated from (3)

$$dv = \frac{V}{P} dp - \frac{Vp}{P^2} dP + \frac{p}{P} dV$$

The differentials can be regarded as the maximum errors in the values measured Then

$$dv_{max} = \pm \left(\frac{V}{P}\left|dp\right| + \frac{Vp}{P^2}\left|dP\right| + \frac{p}{P}\left|dV\right|\right)$$

The relative errors are obtained by dividing by $v = \frac{pV}{P}$ i e (3)

$$\frac{dv_{max}}{v} = \pm \left(\frac{|dp|}{p} + \frac{|dP|}{P} + \frac{|dV|}{V}\right) \tag{4}$$

where V is a substitution for $(\frac{T_b}{T_a} V_a + V_b)$

In equation (4) the relative error in the determination of p is of the order 3 % Relative errors of the same order of magnitude are then permissible for P and V P is about 760 torr and this value will have to be known within 0 03 · 760 torr ∾ 20 torr Thus there is no need to determine the ambient pressure exactly V is about 250 cm³ and will have to be determined within 0 03 · 250 cm³ ~ 8 cm³ V consists of two terms ($\frac{T_b}{T_a} V_a$ and V_b) The latter is as a rule about 8 cm³ This value could almost be omitted and in any case a rough estimate of the volume of the middle ear is quite sufficient In the first term the quotient $\frac{T_b}{T_a}$ was introduced because the tank was kept at room temperature, not at body temperature If this fact is neglected, the error will be $\frac{T_b}{T_a} V_a - V_a \sim 12$ cm³

If, as in the "ear snorkel" article, the size of volume B is neglected and if this volume is normal (8 ml), this approximation results in an error of the determinations of —3 per cent, while the error in reading the recording of the pressure change is ±3 per cent. For this reason the error of the volume passing through the Eustachian tube ranges between 0 % and —6 %. The barometer level P can easily be determined with an error margin of only 0.1 per cent. During "ear snorkel" experiments the pressure *in the measuring system A+B rises by 1 mm Hg owing to inflation through* the tube. According to $v = \frac{p}{P} V$ a volume of $\frac{1.0 \cdot 310}{760 \cdot 298} \cdot 250000 = 340$ mm³ has passed through the tube. Given the above-mentioned limits of error, the correct value in this case lies between 340—360 mm³.

In the article "Controlled Ear Aspiration" and "Clinical Volume Determination" Boyle's law is used. With regard to an unavoidable uncertainty in the instrument readings the real value of the closed manometer-ear space volume and the air volume passing the Eustachian tube will not deviate more than 10 per cent from the experimental value. This error, however, can easily be reduced if necessary.

*

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta OTO-LARYNGOLOGICA

SUPPLEMENTUM 183

PAPERS DEDICATED TO

EELCO HUIZINGA

Professor of Oto Rhino Laryngology
University of Groningen

ON HIS SEVENTIETH ANNIVERSARY
11 September 1963

ACTA OTO-LARYNGOLOGICA KARLAVÄGEN 41, STOCKHOLM Ö

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

Acta
OTO-LARYNGOLOGICA

S U P P L E M E N T U M 183

PAPERS DEDICATED TO

EELCO HUIZINGA

Professor of Oto-Rhino-Laryngology
University of Groningen

ON HIS SEVENTIETH ANNIVERSARY
11 September 1963

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

ACTA
OTO-LARYNGOLOGICA

SUPPLEMENTUM 183

PAPERS

DEDICATED TO

EELCO HUIZINGA

Professor of Oto-Rhino-Laryngology
University of Groningen

ON HIS SEVENTIETH ANNIVERSARY
11. September 1963

GRONINGEN 1963



Foto Harry van der Zwaag

TABLE OF CONTENTS





PUBLICATION OF THESE PAPERS WAS MADE POSSIBLE
BY

Annyas, A P L H
Baarsma, P R
Bakker, R
Bleeker, J D J W
Bleeker, G F
Brune Altes, Joh C de
Burger, D
Damste, P H
Dishoeck, H A E van
Edens, E Th
Gils, J J P van
Graaf, J de
Graaf, W J de
Heege, F H ter
Hoeksema, P E
Hoving, E W
Huizing H C
Iwema, J
Jongert, H
Kruisinga, R J H
Kruizinga, P J W
Leezenberg, J A
Meer H D van der
Meulen P van der
Minne, J M van der
Moolenaar Bijl, A J
Nienhuis, J H
Nienhuis-de Jong, H
Oldenstam, R A
Oostvogel, F E E
Pietersen-German, J L M
Pothoven, W J
Rap, A A
Reyntjes, J A
Rietema, S P
Schierbeek, P
Siemens, B W L
Sinninghe Damste, P J
Smelt, G J
Tan, T S
Taselaar, M
Ubbens, D R
Vettorato, B W
Vis, K van der
Vrolijk, J M
Waalkens, J W M. H
Waning Bolt, G van
Warringa, R J
Weering, IJ F van
Westerbeek, C

The Harmannus Simon Kamminga Foundation

The Dutch Oto-Rhino-Laryngological Society

PUBLISHING COMMITTEE.

J. DE GRAAF

P. E. HOEKSEMA

P. SCHIERBEEK

P. J. SINNINGHE DAMSTÉ

DRUKKERIJ Fa. H. SMIT Azn. GRONINGEN

TO PROFESSOR DR EELCO HUIZINGA FOR HIS 70th BIRTHDAY

The Editors of this volume have asked me to write an introduction about Eelco Huizinga on the occasion of his seventieth birthday and I have to confess that even now I cannot believe that he has reached this age

I have looked up certain facts and, indeed, this young man has been born September the eleventh, 1893, in Semarang on Java

As a boy he went to Groningen to attend school and later the University Being the son of an E.N T man it was not surprising that he followed the same path, became a doctor and an oto-rhino laryngologist

He was a pupil first of Prof Schutter and afterwards of his successor, Prof Benjamins With the character he has inherited from his Groningen ancestors he was bound to be faithful to his town and university

In 1940 he succeeded Benjamins in the chair for Ear, Nose and Throat at the Groningen University, a place he has held very actively and honorably since that time

He succeeded Benjamins also in another important position i e as General Secretary of the Collegium Oto-Rhino-Laryngolocium Amicitiae Sacrum, the inter national society of scientific workers in the domain of Ear, Nose and Throat

In 1926 this Collegium was founded in Groningen and Huizinga was in it from the beginning For twenty years he was the axis and pushing force of the Collegium O R L A S from which position he retired in 1961

Is it really necessary to talk about his deeds and performances? Do I have to tell you that he travelled all over the world to lecture, to teach, to learn but above all to discuss important points with his friends, everywhere in the world?

Is it necessary to tell you about his brilliant scientific work, on the labyrinth, on bronchoscopy, on important clinical, physiological and anatomical problems but also on amusing details like the achievements of the sword swallower?

Must I count all the honorary functions, degrees and orders showered upon him, like his membership of the Royal Dutch Academy of Science, his fellowship of the Royal Society of Medicine in London, of the Royal College of Surgeons in Ireland, of the Swedish Society of Medicine?

Is it of more importance that he is a bearer of the ribbons of Dutch, Belgian and French orders or a honorary or corresponding member of a countless number of ear nose and throat societies in innumerable countries?

No I think this would be history and Eelco Huizinga is by no means a historical figure He may be seventy now but his mind is young He may be member of honour of our Dutch Society, de Nederlandse Keel, Neus en Oorheelkundige Vereniging but he is one of its most active members

He is also an active traveller, still attending all the important *international* meetings where his presence is always of great value on account of his great experience in those matters e g as president of the international Congress in Amsterdam in 1953

I am sure I am forgetting lots of facts but they are not vital The main fact is that Eelco Huizinga was and is a character, a wise man, a humanist undogmatic and open to every rational new point of view He has not lost his charm in seventy years, on the contrary, it is still growing and he is one of the few people, who know how to be a true friend

I have to write these words in the name of the members of the Dutch E N T Society, but I am not able to leave out all my feelings of friendship for Eelco Huizinga and for the companion of his life, Mrs Huizinga, the silent force in the background

Long may they live!

L. B W JONGKEES
President of the Nederlandse Keel Neus en Oorheelkundige Vereniging

SOUND TRANSMISSION IN EARS WITH THREE FENESTRA

Experimental Studies on Sound Transmission in the Human Ear III

by

H C ANDERSEN C C HANSEN and E B NEERGAARD *

Aarhus Denmark

It is the present conception that the effect of the acoustic transmission upon the perception organ encounters deformations of the basilar membrane caused by displacements of the labyrinthine liquid These displacements are reflected in the movements of the round window membrane Conclusively the excursions of this membrane represent not only the physical stimuli of the perception organ but also the overall performance of the sound transmission of the ear

In previous papers we have described a method to record the volume amplitude and the phase of the round window in postmortem human temporal bones Accordance between the physical character of experimentally produced alterations in

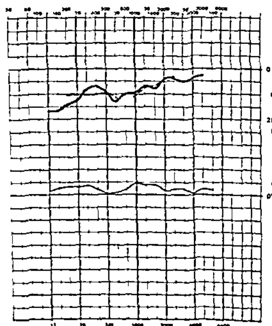


Fig 1 Changes in volume amplitude and phase of the round window as a result of fenestration of the horizontal semicircular canal. Full line specimen no 52 broken line specimen no 37

* *From the Department of Otolaryngology University of Aarhus*

the middle ear and the resulting changes in the movements of the round window has been demonstrated For example it was shown that increase of a stiffness in the middle ear mechanism brought about by pressure changes across the tympanic membrane results in amplitude and phase shifts of the round window vibrations corresponding to increased stiffness of the system"

These experiments were extended to involve examinations of the influence of a third labyrinthine fenester upon the sound transmission The clinical aspect of this question How do ears with three fenestra hear' has been dealt with in publications concerning the hearing in ears with labyrinthine fistula (Leegaard et al) and in ears with mobile stapes after fenestration of the horizontal canal (Gravendeel) In the following we have tried to contribute experimentally to this problem

The representation of the physical stimuli of the perception organ by the displacements of the round window is justified only in conditions where the cochlea is left an unimpaired unit This should be a reasonable assumption also during fenestration as long as the fenester is located on a semicircular canal

Postmortem human temporal bones were used and the volume amplitude and the phase of the round window were recorded during application of a sound pressure of 107 db in the external meatus before and after fenestration of the ampulla of the horizontal semicircular canal

Fig 1 illustrates the effect on the sound transmission caused by the fenestration The low frequency transmission from oval to round window is reduced to 7—17

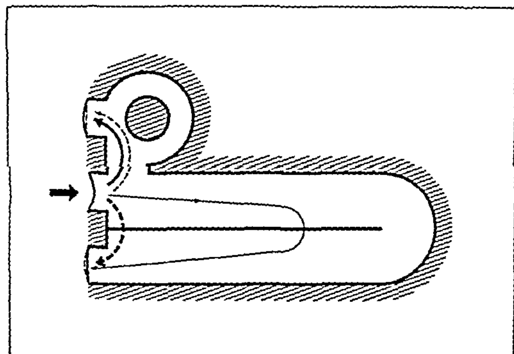


Fig. 2 Diagrammatical representation of the sound paths in the labyrinth after fenestration Full line low frequencies broken line high frequencies.

db below the normal value while at the frequencies above 2000 cps the effect of the acoustic shunt appears to cause less than 5 db transmission loss

The pronounced reduction of the transmission at the low frequencies and the comparatively small influence at the high frequencies appear to be in accordance with the assumption that the third window acts as a shunt impedance with the characteristics essentially of a mass In fig 2 this effect is diagrammatically illustrated indicating the assumed different effective pathways in the labyrinth for the high and the low frequencies It will be seen that the phase shift towards increased phase velocity which has been recorded is also in accordance with this simplified model

REFERENCES

ANDERSEN H C C C HANSEN and E B NEERGAARD 1962 Acta O o-Laryng *54* 511 and Acta Oto-Laryng in print

GRAVENDEEL D W 1957 Acta Oto-Laryng. *48* 433

LEEGAARD TRULS and THOR RÖHRT 1959 Acta Oto-Laryng *50* 42

THE CLASSIFICATION OF FORMS OF COLLAGENOSIS AFFECTING THE EAR, NOSE AND THROAT

by

M. ARSLAN *

Padua, Italy

INTRODUCTION

The inclusion among collagenous diseases — a term which must bear a very wide meaning for the purposes of the present work — of certain otorhinolaryngological diseases must be based on the morphological semeiological and clinical factors

In the present paper we wish to try, for the first time in the literature, to unite all the aspects of the problem in a single group, but to do this we must first of all explain clearly the premises from which we start

It should be unnecessary to point out that whatever conclusions we reach and whatever theories we put forward these cannot be regarded as established facts Our limited knowledge of the pathology of the collagen the polymorphism of the best known clinical pictures of collagenous diseases the looseness of the classification, the variability of the symptoms laboratory findings etc, that are considered specific of these diseases and finally the difference of opinion on the subject, allow us merely to suggest certain methods of approach in studying the problem These reservations must also be made because collagenous diseases affecting the ear, nose and throat are only the local signs of an infection of the collagen which may or may not constitute the first phase of a generalised clinical picture of collagenosis We say clinical and not anatomopathological since, as is well known and as we ourselves have occasion to observe collagenous diseases may be limited to the upper air ways without there being any clinical signs or symptoms at the level of the viscera or of the large organic systems though laboratory tests and histological examination may reveal signs of active generalised infection of the collagen

We must first make a distinction between those otorhinolaryngological manifestations of collagenous diseases which appear in the course of collagenosis that has been active for some time and which we shall call secondary manifestations, and those which are limited and primary as we have mentioned above

The secondary manifestations are represented by the clinical pictures, or sometimes simply the morphological alterations or functional disorders, which one observes for example in the course of lupus erythematosus malignant dermatovisceritis diffuse scleroderma etc

* *Department of Ear Nose and Throat University of Padua (Head Prof M Arslan)*

We shall not discuss these secondary manifestations (whose various aspects have often been described in reports of clinical cases of various collagenous diseases) for the following reasons (1) at the present moment the study of collagenous diseases is concerned, on the clinical plane, with justifying or denying the validity of the classification of particular clinical pictures and therefore has stressed the general aspects of the problem so that a thorough discussion of the secondary manifestations can be postponed till a later date, (2) these secondary manifestations are not always the clinical expression of disorders of the collagenous connective of the upper air ways or of the ear, but are often subsidiary pictures which are not directly connected with the collagenosis but rather the consequence of a weakening of the organism's defences which arises, by an aspecific mechanism, as a result of serious collagenous disease

The study of the primary forms of collagenosis of the otorhinolaryngological area is both interesting and rewarding These forms have an unknown aetiology and present clinical, morphological and histopathological characteristics which are identical with or very similar to those of systemic collagenous diseases and which are considered pathognomonic, within, of course, the limits of our knowledge of the subject If we apply the criteria which various workers in particular MALA GUZZI VALERI, have proposed for defining a collagenous disease (and which everyone accepts), then we shall have to pay particular attention to a large number of manifestations of the upper air ways and ear, including (1) the extreme chronicity of the clinical picture, (2) its evolution in a series of attacks, (3) the fact that the disease maintains itself, (4) its slightly infectious aetiology, (5) the isopathological aspects of chronic granulomatosis with clear signs of damage to the collagenous connective, and (6) the fact that there are almost always signs of dysreactivity either in the histopathological pictures of the lesion or, more rarely, in the findings of laboratory tests, etc

One can in fact say the disease can present innumerable clinical pictures of ulcerative, proliferative, desquamatous, oedematus, dystrophic and dysendocrine lesions, of whose aetiology and pathogenesis we know nothing It is quite justifiable, therefore, to try to classify these pictures as collagenous diseases or, in a wider sense, as forms of mesenchymopathy But it is clear that certain limitations must be set, as KLEMPERER and CAVALLERO have warned

Special attention must be payed to the contributions that our department has made to the study of collagenous diseases involving the ear, nose and throat Our investigations have covered the histopathology, semeiology, clinical picture and treatment We have tried to coordinate the research being done by our various assistants so as to give some unity to the work as a whole Thus, we began to use arteriography of the external carotid for diagnosis after our histopathologist had succeeded in identifying signs of vascular damage in cases of definite collagenosis The results of this team work were communicated to the International Congress of Otorhinolaryngology held in Washington and the Congress of the Italian Society of Otorhinolaryngology held in Pisa (ARSLAN, RICCI, DE STEFANI, BOTNER, SALA, MEGIGHIAN and FIORETTI, 1957)

We must list the otorhinolaryngological diseases which we have studied and defined as actual or presumed collagenous diseases

In an earlier paper (1954) we turned our attention on the following six diseases 1) malignant granuloma, 2) amyloidosis, 3) sarcoidosis, 4) scleroderma, 5) simple ethmoidal polyposis without inflammation, and 6) ozena After a further study of the various aspects of the problem, we now consider that we can exclude sarcoidosis (Boeck's sarcoid) and add three other diseases 1) Gourgerot Sjogren's syndrome, 2) collagenosis of the auricular pavilion and of the nasal pyramid, a primary or secondary form of rheumatoid arthritis, and 3) focal infection

These two lists are not to be considered in any way final Future studies may well lead to the elimination of some diseases and the addition of others

LES VARIATIONS DE LA RÉPONSE DU NERF COCHLÉAIRE CHEZ LE COBAYE, EN FONCTION D'UNE STIMULATION SONORE ITERATIVE PROLONGÉE

par

M AUBRY, M BURGEAT et P. PIALOUX
Paris, France

I INTRODUCTION

L'activité de la plupart des fibres nerveuses en provenance des récepteurs périphériques peut subir des variations d'ordre quantitatif dans certaines conditions de stimulation Adaptation, inhibition, fatigue sont autant de facteurs agissant sur le nombre des influx par fibre, ce qui, pour un groupe de nombreuses fibres implique une diminution d'amplitude du potentiel d'action Le nerf cochléaire n'échappe pas à cette regle

Un certain nombre d'experimentateurs (Galambos[1], Fex[2]) ont permis de demontrer qu'il était possible d'agir sur l'amplitude des potentiels d'action auditif en stimulant electriquement des structures nerveuses supérieures

Mais est il possible d'obtenir une variation d'amplitude du potentiel d'action auditif sans utiliser les artifices difficilement controlables d'une stimulation électrique des voies centrales? Un stimulus sonore peut il créer à lui seul des modifications du potentiel d'action dont il est l'origine?

Alt'man[3] repond en partie a notre question Il relate en effet les résultats d'expérimentations effectuées chez le Chat au cours desquelles il a démontré que la chute d'amplitude du potentiel d action que l'on peut obtenir au cours d'une stimulation sonore tres prolongee peut être empêchée si l'on effectue l'ablation de l'aire auditive controlaterale

Il n'est certes pas nécessaire d insister sur le grand interêt d'une telle démonstration Néanmoins, avant de faire intervenir des structures très complexes dont il est encore fort difficile d apprecier le role avec exactitude, il nous a paru intéressant de determiner dans quelles limites un stimulus sonore est capable d'engendrer des variations d'amplitude du potentiel d'action auditif et d etudier ces modifications en fonction des potentiels cochleaires connus

Nous avons entrepris une série d expériences au cours desquelles nous nous sommes particulièrement attaches à determiner le mode de stimulation auditive ainsi qu a comparer les potentiels microphoniques cochléaires et les potientiels d'action enregistrés simultanément par des electrodes intracochleaires

II TECHNIQUE

II 1 Il a ete nécessaire de respecter deux importantes conditions avant d entreprendre l experimentation

We must list the otorhinolaryngological diseases which we have studied and defined as actual or presumed collagenous diseases

In an earlier paper (1954) we turned our attention on the following six diseases 1) malignant granuloma, 2) amyloidosis, 3) sarcoidosis, 4) scleroderma, 5) simple ethmoidal polyposis without inflammation, and 6) ozena After a further study of the various aspects of the problem, we now consider that we can exclude sarcoidosis (Boeck's sarcoid) and add three other diseases 1) Gourgerot Sjogren's syndrome, 2) collagenosis of the auricular pavillion and of the nasal pyramid, a primary or secondary form of rheumatoid arthritus, and 3) focal infection

These two lists are not to be considered in any way final Future studies may well lead to the elimination of some diseases and the addition of others

etait mise en place dans le tour de spire basal de la cochlee L electrode était maintenue contre la bulle a l aide de ciment

Une tracheotomie etait pratiquee pour la mise en place d une canule destinee a la respiration artificielle

L animal etait ensuite place sur une table stereotaxique de Horsley-Clark situee dans une cage de Faraday

Le stimulus sonore etait dirige vers les oreilles a l aide d un tuyau en matiere plastique s adaptant aux tiges a oreilles creuses de contention L enregistrement était effectue apres preamplification sur un oscilloscope dont la trace etait photographiee

Apres verification de l enregistrement des deux cochlees l animal etait curarise (Flaxedil) et on reliait sa canule de tracheotomie a la Pompe a respiration

Le son test etait ensuite envoye alternativement aux 2 oreilles jusqu a ce que la surveillance des reponses cochleaires permettent de verifier que la periode de stabilite etait atteinte avec certitude

Les reponses cochleaires (MC PA) etaient alors mesurees et photographiees Puis le stimulus prolonge etait ensuite emis dans une oreille pendant 60 minutes (*oreille testee*)

A la fin de cette période on effectuait une nouvelle mesure des potentiels cochleaires des deux oreilles a l aide du son test (oreille testee oreille temoin) Un enregistrement photographique etait fait 3 minutes apres la cessation du son prolonge

Pendant toute la duree de l experience

la temperature de l animal etait surveillee ainsi que le rythme cardiaque enregistre a l aide d electrodes thoraciques

III RÉSULTATS

III 1 L oreille temoin qui n a pas reçu de son prolonge n a montre aucune modification significative des potentiels microphoniques et des potentiels d action

III 2 Au niveau de l oreille testee on a constate apres une heure de stimulation prolongee que si les potentiels microphoniques cochleaires etaient restes stables par contre les potentiels d action avaient montre une nette diminution

III 3 Si l on etablit le rapport des amplitudes des potentiels d action des oreilles testees avant et apres la stimulation prolongee on obtient une chute de 1 71 alors que les potentiels d action des oreilles temoins ainsi que les potentiels microphoniques des oreilles testees et temoins etaient de l ordre de l unite

IV DISCUSSION

IV 1 Etant donne la stabilité des potentiels microphoniques cochleaires on peut conclure que les variations d amplitude des potentiels d action sont independantes de l activite de l organe de Corti et ne semblent dependre que de structures nerveuses plus elevees

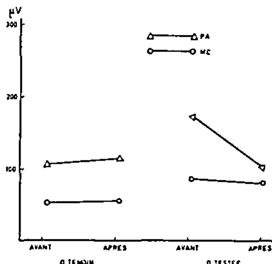
IV 2 La nature de ces variations est plus discutable Neanmoins si nous nous referrons aux resultats d Alt man il semble qu il doive s agir d une inhibition

IV-3 Le premier lieu ou l'action inhibitrice pourrait être envisagée serait la synapse organe de Corti - fibre afferente

Le systeme de fibres efferentes (faisceau olivo-cochleaire de Rasmussen) pourrait en etre la voie d'action sans pour cela sous-estimer le role des structures plus elevees (formation reticulaire, cortex)

Il pourrait, en definitive, s'agir d'une reaction des centres vis à vis d'une stimulation prolongee ayant perdu tout caractere informatif

Nous poursuivons actuellement nos experiences en vue de déterminer le rôle des structures nerveuses centrales sur cette inhibition Il s'agit là d'un phénomène dont la connaissance serait du plus haut interet, tant en ce qui concerne la physiologie de l'audition, que les consequences physio-pathologiques que l'on pourrait en deduire



V CONCLUSIONS

V 1- Il existe normalement chez le Cobaye une diminution des potentiels d action auditifs pour une stimulation sonore itérative et prolongee, non traumatisante

V 2 L'origine invoquee est celle d'une inhibition dont serait responsable le faisceau de fibres efferentes de Rasmussen lui meme placé sous la dependance de structures nerveuses plus elevees

BIBLIOGRAPHIE

1 GALAMBOS R Suppression of auditory nerve activity by stimulation of efferent fibres to cochlea
J Neuro-Physiol 1956 19 424-437

2 FEX J Augmentation of cochlear microphonic by stimulation of efferent fibres to the cochlea
Acta O[illegible] La[illegible] 1959 50 540-541

3 ALT[illegible]A [illegible] Le r[illegible]e de l'ecorce auditive dans la repression de l'impulsion afférente dans [illegible] l[illegible]s d'une stimulation sonore prolongee
Com[illegible] [illegible]ca[illegible]e des Sciences de l'U R.S.S 1961 136 500-503

RECHERCHES SUR UNE VENTRILOQUE NÉERLANDAISE

by

Jw VAN DEN BERG

Groningen, Pays Bas

„Recherches sur un ventriloque neerlandais" fut le titre d'un article que le professeur Eelco Huizinga publia, il y a plus de 30 ans, en 1931, au sujet de ses recherches sur l'artiste „le Professeur Barandini" qui fut a l'epoque le meilleur ventriloque neerlandais

A l'occasion du jubile du professeur Huizinga, il nous semblait valoir la peine de reprendre ces recherches et, si possible, de les completer à l'aide des appareils modernes „Le Professeur Barandini" est mort entretemps Pour le moment Madame Lahnstein est sa meilleure eleve (voir figure 1), elle a consenti a y preter son concours



Fig. 1 Position de la bouche de Mme Lahnstein pendant la ventriloquie

Commençons pourtant par resumer les plus importants resultats des recherches du *professeur Huizinga* „Le *ventriloque parle* comme dans le cas normal par une formation exclusivement expiratoire de la voix, mais l'air est plus comprimé par une stenose Ce rétrecissement se produit par le fait que l entree du larynx se trouve fortement contractee Dans ce but, la basse de la langue et l'epiglotte se reportent tres fortement en arriere de façon que l epiglotte s'applique contre la paroi postérieure du larynx' „Durant la ventriloquie le larynx est encore tiré un peu vers le haut contre la base de la langue La voix du ventriloque est une voix etouffee La consommation d air est beaucoup moindre que dans la voix normale", „ le ventriloque parle presque continuellement avec une position inspiratoire maximale du larynx Le diaphragme s abaisse et reste continuellement fixe dans une position tres basse Le thorax est fortement dilaté " „ le diaphragme est fortement mis en jeu L epigastre est reporte en avant, le ventre est fortement

gonfle tout le temps de la ventriloquie dans la position d inspiration ce qui donnait anciennement l illusion que la voix se produisait par inspiration Il se peut que la cooperation reelle du ventre ait contribue à la creation du mot ventriloquie

A la main d une grande serie de radiographies que Huizinga fit le premier de la ventriloque il conclut Le son etouffe particulier qu on perçoit dans la ventriloquie se produit donc parce que l on parle avec la position maximale d inspiration du thorax avec une position du larynx analogue à celle quand on presse

Il fut ensuite premier a enregistrer les vibrations du domaine trachelien causées par les vibrations du larynx et il donna une excellente description de la production des voyelles et des consonnes des difficultes se presentant devant les differents phonèmes du fait que la bouche et la gorge ne bougent pas enfin la façon grace à laquelle l illusion se produit

Nous avons etudie la ventriloquie de Madame Lahnstein de la manière suivante 1 Enregistrement simultane du signal sonore (phonique) et des vibrations du cou juste au-dessus du larynx suivi d une analyse a l aide du Sonagraphe 2 des radiographies laterales du cou 3 des radiotomogrammes du larynx L aperçue que Huizinga donne des resultats sur son sujet masculin le Professeur Barandini correspond à ce que l on accepte quasi unanimement aujourd hui mais ne s applique pas sans autre a l eleve feminin de Barandini Madame Lahnstein C est qu elle se sert de deux voix ventriloques une voix haute qu on attribue à une charmante poupee du sexe feminin et une voix basse appartenant à un polisson l enfant terrible

Dans les deux genres de voix elle dispose à peu près d une octave et les vo x se produisent apparemment sans la moindre peine et avec un grand volume Chez elle il n est pas question d une contraction extreme comme chez les artistes masculins Elle evite sciemment la contraction extreme afin de ne pas forcer le larynx

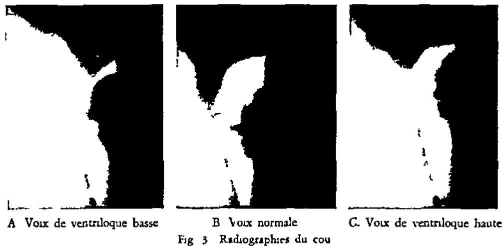
Il ressort des figures 2 et 3 que la voix ventriloque basse se produit dans le registre de poitrine la voix normale dans le registre mixte et la voix ventriloque haute dans le registre de fausset Ces experiences sont en parfait accord avec les



A Vo x [illegible] a se B Vo x normale C Vo x de ven lo e hau e

F g Rad otomog ammes du larynx

resultats des analyses a l'aide du Sonagraphe, voir figure 4 L'on a donc ici l'exemple d'une ventriloque qui, d'une façon parfaite, est a meme de se servir des registres normaux de sa voix pour excercer sa profession



A Voix de ventriloque basse B Voix normale C. Voix de ventriloque haute

Fig 3 Radiographies du cou

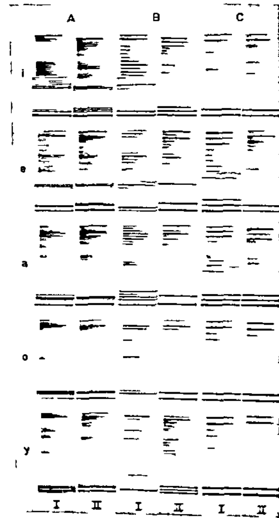


Fig. 4 Analyses avec le „Sonagraphe" A Voix de ventriloque basse B. Voix normale C. Voix de ventriloque haute I Vibrations de l'air II Vibrations du cou

LA PRESBYACOUSIE

par

F. CRABBÉ

Bruxelles, Belgique

Dès le premier âge l'ouïe subit une involution progressive dont les premiers signes se marquent déjà nettement vers l'âge de 20 ans. Cette involution, semblable aux deux oreilles, s'affirme sous forme d'une pente descendante sur les sons aigus.

Quant aux sons graves, ils sont touchés de façon diverse mais toujours dans une mesure moindre. Chez certaines personnes âgées on note parfois un Rinne audiométrique légèrement négatif sur les sons graves.

Dans toutes les atteintes de l'oreille interne se remarque une difficulté à comprendre la voix parlée surtout dans un bruit ambiant. Mais chez la personne âgée cette perte de discrimination est infiniment plus accusée que chez le sujet jeune, hors de proportion avec l'audiogramme tonal (Pestalozza et Shore).

Ces faits, connus depuis longtemps, restent interprétés de manière diverse.

Certains (Hofer, Whright, Yannoulis, etc.) en cherchent la raison dans la rigidité croissante du système de conduction.

Mayer a décrit une rigidité de la membrane basilaire surtout accusée dans la portion proximale du tour de spire basal.

Wittmaack insiste sur l'hypotonie du canal cochléaire allant jusqu'au collapsus.

Toutes ces altérations, rendant le système oscillant plus rigide, en augmentent l'impédance. Elles peuvent éventuellement expliquer une diminution d'ouïe pour les sons graves et un déplacement de la fréquence de résonnance, mais elles ne permettent pas de comprendre les caractères principaux de la courbe audiométrique tonale et vocale.

Au niveau de l'oreille interne évoluent deux processus complètement indépendants de l'avis de tous les auteurs (Wittmaack, von Fieandt et Saxen, Crowe, Guild et Polvogt, Schuknecht).

Chez tous les vieillards on note une atrophie du ganglion de Corti où les neurones diminuent progressivement en nombre jusqu'à extinction. Cette atrophie débute à un âge avancé à l'extrémité proximale de la spire basale et progresse très lentement en direction distale. Les filets nerveux correspondants s'atrophient et disparaissent. La cochlée membraneuse peut rester parfaitement indemne au cours de cette évolution. Dans le cas de Schuknecht le rameau efférent de Rasmussen était resté normal.

Contrastant avec cet appauvrissement neuronique d'apparition très tardive et d'évolut[illegible]n extrêmement lente s'amorce dès l'âge moyen une atrophie de l'épithélium du canal [illegible]chléaire, elle aussi progressive, qui atteint toutes les composantes du système. Le processus débute au niveau de la strie vasculaire et de la proéminence [illegible]uant la sécrétion d'endolymphe, d'où abaissement de la membrane [illegible]lant jusqu'au collapsus et à sa soudure avec la strie et l'organe de Cor

Le limbe spiral est atteint de la même manière, entraînant l'altération du gel instable de la tectoria dont il est l'organe nourricier

La papille de Corti avec les cellules ciliées, nourrie uniquement par l'endolymphe, subit le même sort Les premières lésions se marquent au niveau du tissu de soutien, aboutissant à l'écroulement du tunnel Au cours de cette évolution les cellules ciliées diminuent en nombre, leurs cils tombent En fin de compte la papille disparaît

Les neurones du ganglion de Corti participent à ce processus avec leurs filets nerveux Schuknecht relève l'atrophie concomitante du rameau de Rasmussen

Pour von Fieandt et Saxén, les foyers dégénératifs s'établissent, sans aucune topographie précise, là où le système vasculaire est altéré et la sécrétion endolymphatique en défaut Pour Guild et al et surtout pour Schuknecht, dont l'avis, basé sur des fixations intravitales, laisse peu de doutes, la dégénérescence s'installe à l'extrémité proximale de la spire basale et évolue en direction distale Elle serait dépendante du processus dégénératif généralisé du tissu mésenchymateux

Ces altérations épithéliales et neuroniques expliquent aisement la courbe d audiométrie tonale

Pour Crowe, Guild et Polvogt, une chute d'ouïe lentement progressive sur les aigus irait „grosso modo" de pair avec la dégénérescence du ganglion de Corti, tandis qu'une chute abrupte sur les aigus se retrouverait en cas de dégénérescence de l'organe sensoriel

Ce rapport n'est sûrement pas constant en ce qui concerne la lésion ganglionnaire Le cas de Schuknecht montrait une chute brusque au delà de 1 000 c/s et, dans les neurinomes de l'acoustique, on retrouve des audiogrammes d'allure diverse mais qui, tous, presentent une atteinte prédominante des aigus

La recherche du recrutement est rendue difficile du fait de la bilatéralité des lésions et du ralentissement psychique du sujet Tandis que Harbert et Sataloff ainsi que Schuknecht nient l'existence de recrutement dans la presbyacousie, Pestalozza et Shore relèvent 20 % de recrutement presque complet et 30 % de recrutement partiel, en employant la technique de Reger

Un recrutement complet paraît difficilement concevable, l'atrophie du ganglion cochleaire ayant éte note dans tous les cas Mais il paraît logique d'admettre un recrutement partiel d'importance variable, rendu probable du fait d'un seuil auditif *très précis et de l'intensification rapidement intolerable au delà de ce seuil*

Mais le fait le plus gênant pour le vieillard est de ne plus etre à même de suivre une conversation dans le brouhaha Même en chambre silencieuse, sa faculté de discrimination est etrangement amoindrie Si une diminution de la discrimination résulte de l'atteinte de l'oreille interne et de l'existence éventuelle de recrutement, il n en reste pas moins que certaines personnes d'âge se plaignent de cet amoindrissement alors que leur audiogramme tonal est peu altéré

La degenerescence neuronique explique en partie ce phénomene. Si, d'apres Schuknecht, 75 % des neurones d'une portion determinée du ganglion de Corti peuvent disparaître sans qu'en soit affecte le seuil auditif des fréquences correspondantes, il n'en va pas de même pour la transmission des signaux complexes de la parole

Goetzinger et Proud rapportent un cas de neurinome de l'acoustique dont l'audiogramme tonal était presque normal tandis que la discrimination vocale était fort diminuée.

Mais la dégénérescence sénile du ganglion de Corti n'est qu'un élément d'un processus d'involution atteignant le système nerveux tout entier et réduisant les facultés intellectuelles du vieillard.

Brody a relevé dans la circonvolution temporale une diminution du nombre de neurones allant jusqu'à la moitié du chiffre normal, thalamus et ganglions gris de la base étant eux aussi entrepris.

L'état des voies acoustiques centrales n'est pas complètement connu à ce jour, mais récemment Kirikae a montré l'appauvrissement des noyaux cochléaires. Par ailleurs la dégénérescence du rameau de Rasmussen, démontrée par Schuknecht, prouve l'entreprise de l'olive et confirme l'atteinte du tronc cérébral.

La diminution du nombre de neurones explique la régression de la faculté de combler les insuffisances de la réception cochléaire.

L'involution des voies cochléaires efférentes prouvée par la dégénérescence du rameau de Rasmussen supprime la faculté d'isoler au milieu d'un complexe sonore le faisceau sur lequel se porte l'attention.

Bocca s'est appliqué à cet aspect du problème en étudiant la faculté de discriminer des mots déformés. Le pourcentage d'intelligibilité est pratiquement le même chez le vieillard et chez le sujet atteint de tumeur du lobe temporal. Bocca en conclut que la perte de discrimination du vieillard est d'origine corticale.

Goetzinger et Roussy ont repris le problème en comparant l'intelligibilité de mots aisés et de mots difficiles à comprendre. Même chez les jeunes il existe une différence. Si la perte de discrimination du vieillard est d'origine corticale, cette différence d'intelligibilité doit croître proportionnellement avec l'âge et uniquement aux dépens de la liste des mots difficiles. En fait il en est ainsi dans une certaine mesure. Mais l'intelligibilité diminue pour les deux groupes de mots et la différence relative ne croît que légèrement. Si l'augmentation de l'écart entre l'intelligibilité des deux listes prouve l'intervention d'un facteur cortical, la perte globale de la faculté de discrimination prouve l'action d'autres facteurs plus périphériques résultant des lésions de dégénérescence étendues à tous les niveaux de l'appareil auditif.

La presbyacousie est donc le résultat d'une involution de l'appareil auditif tout entier comprenant la sclérose des formations de l'oreille moyenne et du labyrinthe membraneux, l'atrophie de l'épithélium sensoriel, l'atrophie du ganglion de Corti, témoins de l'involution généralisée du système nerveux et comprenant les voies acoustiques centrales jusqu'au cortex ainsi que les voies efférentes nécessaires à la concentration de l'attention auditive.

L INVENTAIRE DE LA PHONIATRIE PRATIQUE

par

P H DAMSTÉ *

Utrecht Pays Bas

La contribution medicale au traitement des troubles de la voix et de la parole est fournie pour la majeure partie par l Oto Rhino-Laryngologie et par la Psychiatrie La premiere s adresse aux troubles organiques de l appareil vocal l autre aux phenomenes psychopathologiques qui se traduisent par des troubles de la voix et de la parole

On pourrait dire que ces deux domaines se completent d une façon magnifique Mais ce n est pas vrai Ce sont là deux domaines de la medecine mais pour le medecin ce sont deux aspects d un meme malade Cependant le traitement doit etre conduit par une seule personne et c est la la tache du phoniatre Pour l oto-rhino-laryngologue il est tres difficile de traiter l aspect psychopathologique tandis que pour le psychiatre il est souvent impossible de juger dans quelle mesure l aspect organique entre en jeu

De nos jours il n y a guere de service O R L qui ne se soit attache les services d un logopediste pour traiter les troubles de la voix et de la parole

Il y a cependant lieu de constater que sur le plan medico-scientifique la participation effective dans le traitement est encore assez limitee

La phoniatrie en tant que specialisation medicale exclusive et reconnue n existe qu en quelques pays Aujourd hui la tache de la phoniatrie consiste a integrer tout ce que la medecine peut apporter comme contribution au traitement des troubles de la voix et de la parole et a maitriser ce domaine dans son ensemble Or ceci exige une orientation plus etendue en dehors de la medecine et qui ne soit pas limitee a la seule phonetique

En ce qui concerne la voix pour pouvoir juger ce qui est anormal il faut que le phoniatre connaisse bien les possibilites et les limites de la voix normale Personne ne pourra pretendre qu il suffit pour cela de connaitre a fond la physiologie du larynx

La theorie et surtout la pratique de la reeducation sont indispensables pour connaitre les possibilites et les limites de la voix et discerner les mauvaises influences qui jouent un role Le fonctionnement de l appareil vocal n est en rien comparable au fonctionnement des reins ou du foie independamment de l etat de l organe lui meme son fonctionnement depend directement de la façon dont le sujet en fait usage Or il s agit la d un probleme qui doit etre traite a la fois sur le plan psychologique et sur le plan pedagogique

La therapeutique respiratoire qui est la clef de la reeducation de la voix est

* *De la Clinique O R L (Prof Dr P G Gerlings) de l Universite d Utrecht*

Goetzinger et Proud rapportent un cas de neurinome de l'acoustique dont l'audiogramme tonal était presque normal tandis que la discrimination vocale était fort diminuée

Mais la dégénérescence sénile du ganglion de Corti n'est qu'un elément d'un processus d'involution atteignant le système nerveux tout entier et réduisant les facultés intellectuelles du vieillard.

Brody a relevé, dans la circonvolution temporale, une diminution du nombre de neurones allant jusqu'à la moitié du chiffre normal, thalamus et ganglions gris de la base étant eux aussi entrepris

L'état des voies acoustiques centrales n'est pas completement connu à ce jour mais, recemment, Kirikae a montré l'appauvrissement des noyaux cochléaires Par ailleurs la dégénérescence du rameau de Rasmussen, démontrée par Schuknecht, prouve l'entreprise de l'olive et confirme l'atteinte du tronc cérebral

La diminution du nombre de neurones explique la régression de la faculté de combler les insuffisances de la réception cochléaire

L'involution des voies cochléaires efférentes prouvée par la dégénérescence du rameau de Rasmussen, supprime la faculté d'isoler au milieu d'un complexe sonore le faisceau sur lequel se porte l'attention

Bocca s'est appliqué à cet aspect du problème en étudiant la faculté de discriminer des mots déformés Le pourcentage d'intelligibilité est pratiquement le même chez le vieillard et chez le sujet atteint de tumeur du lobe temporal Bocca en conclut que la perte de discrimination du vieillard est d'origine corticale

Goetzinger et Roussy ont repris le probleme en comparant l'intelligibilité de mots aisés et de mots difficiles à comprendre Même chez les jeunes il existe une différence Si la perte de discrimination du vieillard est d'origine corticale, cette différence d'intelligibilité doit croître proportionnellement avec l'âge et uniquement aux depens de la liste des mots difficiles En fait, il en est ainsi dans une certaine mesure Mais l'intelligibilité diminue pour les deux groupes de mots et la différence relative ne croit que légèrement Si l'augmentation de l'écart entre l'intelligibilité des deux listes prouve l'intervention d'un facteur cortical, la perte globale de la faculté de discrimination prouve l'action d'autres facteurs plus périphériques résultant des lesions de dégénérescence étendues à tous les niveaux de l'appareil auditif

La presbyacousie est donc le résultat d'une involution de l'appareil auditif tout entier comprenant la sclérose des formations de l'oreille moyenne et du labyrinthe membraneux, l'atrophie de l'épithelium sensoriel, l'atrophie du ganglion de Corti témoin de l'involution généralisée du système nerveux et comprenant les voies acoustiques centrales jusqu'au cortex ainsi que les voies efferentes nécessaires à la concentration de l'attention auditive

egalement le degre d intelligence le milieu les exercices les notions du langage et l etat des machoires et de la dentition

Ces examples mettent suffisamment en evidence qu au temps ou la phoniatrie etait en train de s elaborer en l O R L une sorte de phenomene de boutonniere s est presente Apres avoir traverse l enceinte de l O R L elle a pu se developper largement sur d autres terres fecondes

En jetant un coup d oeil sur le champs de travail de la phoniatrie on peut conclure que pour les recherches experimentales un choix étendu d instruments et d appareils est indispensable Pour la pratique au contraire l inventaire se compose de l equipement normal complete par une oreille fine expérimentee et toujours *aux ecoutes*

SUMMARY

Examples of the widely different aspects of voice and speech defects are mentioned The specialty of phoniatrics integrates them and thus prevents the patient being divided between specialties and not being treated as a whole

For the phoniatric practice a capacity for keen listening with experienced ears is more important than a set of instruments

BIBLIOGRAPHIE

BRODNITZ F Vocal Rehabilation (Rochester U.S.A 1959)

DAMSTE P H and HUIZINGA E. Stroboscopic fixation of the vocal cords Pract Oto-Rhino-Laryng 21 349 453 (1959)

SCHMITT J L Atemheilkunst 3e Aufl (Bern/Salzburg 1956)

VIRAL INFECTION IN TWO CASES OF SUDDEN PERCEPTIVE DEAFNESS

by

H A E VAN DISHOECK *

Leyden, The Netherlands

In the last 8 years we observed over 150 patients who became deaf on one or both ears more or less suddenly and dramatically within some minutes or some days Luckily only some of them remained totally deaf or needed a hearing aid Partial recovery was seen often and total recovery of one or both ears incidentally In most cases dizziness and tinnitus were present, often drawing the attention of the patient to his one sided deafness

Already in 1955 we mentioned viral infection as the probable cause of this illness in part of the patients Indeed deafness following mumps and measles and the disastrous effect of purpura on the foetal ear are wellknown. However the fact that other viruses might accidently cause deafness too and even in patients who are only slightly or not at all ill, is not wellknown

Too often etiology and pathogenesis are confused in this connection According to us apart from viral infection the etiology may also be general vascular disorders, Meniere disease, allergy, mental stress, blood diseases, trauma etc , and in all these causes the pathogenesis must either be oedema, thrombosis, blood sludging, spasm or neuritis Occasionally chilling of the ear is reported as the cause of sudden deafness, but most probably this is only a provocation, the cause being f i a latent viral infection

Strong motives and even convincing proof of sudden perceptive deafness being caused by viral infection may be drawn from several observations

1 The existence of an „infectious" illness as a cold, flu, benign meningitis or encephalitis
2 The demonstration of a pathological virus in the throat, the blood, the faeces or the cerebrospinal fluid
3 The demonstration of viral antibodies in the blood

Among our patients more than one third suffered from a mild to a severe cold at the onset of their deafness In some cases lymphocytic meningitis was present, proved by lumbar puncture and in a number of those who came to us in an early stage of their illness a pathological virus could be cultured or the contact with such a virus could be serologically established These investigations were done at the department of Prof Verlinde and Prof Dinger, however of course for a restricted number of viruses Positive results were obtained for influenza, Columbia S K Coxackie and mumps It must be mentioned that in the many cases of high and later decreasing positive titres for mumps, a mumps parotitis was

* *From the Department of Oto Rhino-Laryngology, University of Leyden (Director Prof Dr H A E van Dishoeck)*

present only in one patient, whereas nearly all the others had had mumps previously Here reinfection must thus be considered to have caused an „anamnestic" reaction in the cochlea

Recently we observed two patients who are of special interest for the clinical picture of viral perceptive deafness In the first patient viral infection is proved, in the second one very probable

The first patient, a female, aged 55, became acutely ill on December 5th, 1961, with rigors and cough Temperature 40°C She developed symptoms of bronchitis She was given 500 m gram phenoxymethylpenicillin per day December 12th a dullnes appeared over the lower lobe of the right lung The temperature remained high On this day the patient felt very ill, was somnolent and the signs of lung inflammation worsened She was hospitalised on December 12th in the Department of Internal Medicine of the University Hospital, Leyden. (Prof Dr J Mulder)

Physical examination The patient had an irritating cough and produced a small quantity of sputum She had no headache She was slightly cyanosed Temperature 39°C Pulse 100 Blood pressure normal Over the right lower lobe a soft tubular breathing sound was present, besides many fine crepitating rales *Laboratory* Hb 76 pCt, white cells 8200 mm^3 Eosinophils $10/mm^3$ R C S R 81/105

X ray lungs shadow in lateral segment of right middle lobe and in the basal segments of right lower lobe Blood culture sterile Gram stain of sputum and culture no bacterial flora Cytological smear of sputum many leucocytes, no degenerated apithelial cells

The patient was treated during only 1 day with 1 million units penicillin G and 1 gram streptomycin per day

On December 14th tetracyclin treatment was started, 2 gram per day during 8 days, because the diagnosis of primary atypical pneumonia seemed probable

On December 16th the temperature became normal and gradually she improved.

The right lung cleared gradually and the X ray became normal on Janurary 23th 1962 The titre of cold agglutinin rose from 1 8 to 1 64, and the antibody content of the serum against the Eaton agent, tested in chicken embryo using fluorescent labelled antibody, rose from 1 80 tot 1 640 (Dr J F Ph Hers, Department of Internal Medicine) There was no rise of titre in the C.F test against influenza virus A and B, adenovirus and psittacosis

Ear complication On December 22th, 17 days after the beginning of her illness and one day after stopping the tetracyclin, the patient suddenly complained of deafness in the left ear with a little pain during the first day and a slight tinnitus but no dizziness Vomitus during some days The tympanic membranes appeared normal

Audiometrical examination Left ear perceptive deafness of 70dB for low tones Above 1200 Hz total deafness Recruitment positive Tone-decay test negative Right ear normal.

According to the neurologist (Dr W Kramer) the somnolence and following disturbance of the rhythm of sleep, an anisocoria, a slight spontaneous nystagmus and slight abnormalities in the electro-encephalogram point to a mild cerebral reaction

Her son living with her suffered at the same time from viral pneumonia and temporary deafness On audiometrical examination on 5 January 1962 the hearing proved to be normal again It is not possible to establish if he suffered from perceptive deafness at the time of his illness

Epicrisis Proved viral infection (pneumonia, encephalitis) in mother and son complicated a remaining perceptive deafness in the mother and total recovery of a deafness of unknown localisation in the son The mother received one day 1 gram of streptomycin

Second patient Cath J van W, female 19 years old.

On November 13th 1961 bilateral mastoidectomy was performed elsewhere after bilateral otitis externa (bact alkaligenes faec.) complicated by mastoiditis and severe deafness She was treated with pen 1 million units and streptomyc 1 gram and later on with terramyc. 1½ gram, without any effect on the fever or the clinical picture On December 13th 1961 she was admit

ted to our clinic because of this bilateral deafness, severe ear ache needing morphia bilateral otitis externa and right sided facial paralysis By exploration of both mastoids it was found that they were filled with whitish tissue and a watery brownish fluid *Histology* chronic inflammation with round cell infiltration and foreign body reaction resembling in some parts granulation tissue *Bact* Cultures from mastoid and external meatus negative t b neg, moulds neg, virus cultures as far as done neg Temp 39°, R C S R 40/64 White cells /mm³ 8000 — leuco 75% Lymph and Mono 25%

Audiogram R total deafness, L perceptive deafness of 50dB

January 4th 1962 general condition much improved R C S R 2/5 Temp normal R ear total deafness L ear air conduction 50dB, bone conduction 30dB hearing loss Facialis R total paralysis February 5th facialis decompression followed by prompt recovery March-April R ear total deafness No vestibular reactions L ear air conduction 50dB, bone conduction 10dB hearing loss The patient has been rehabilitated with a hearing-aid

Epicrisis Otitis externa, media, mastoiditis and facial neuritis without any bacterial infection Antibiotics without any effect Atypical lymphocytic mastoid infiltration R ear total perceptive deafness, L ear recovery of a perceptive deafness but remaining of transmission deafness probably caused by adhesions Presumably this atypical ear inflammation is caused by an undetected virus

Discussion Myringitis as a complication of viral infection e.g in influenza is wellknown The same holds true for benign lymphocytic meningitis and encephalitis Damage done by viral infection to the middle ear is not familiar to the otologist



Microphotograph shows ear of a patient who experienced sudden deafness Right ear was normal, left ear diseased Note shrinkage of tectorial membrane, organ of Corti and stria vascularis There was also a total loss of hair cells in saccule (By courtesy of Dr Schuknecht)

Recently Schuknecht c s examined the temporal bones of four patients with unilateral sudden deafness of unknown etiology According to him the changes are similar to those which occur in human labyrinthitis of known viral etiology and are unlike the pathological changes resulting from experimental vascular occlusion in animals He also notes, as we do, that the evidence suggests that mumps or a related virus may be an important etiological factor in sudden perceptive deafness

SUMMARY

Viral infection and reinfection with the mumps virus is probably an important cause of sudden perceptive deafness The damage done by this infection may be restricted to the cochlea but may also involve the brain and may also cause severe sterile inflammation of the external ear, inner ear and mastoid Two patients are reported one suffering from viral pneumonia and sudden deafness and the other from severe atypical mastoiditis myringitis and facial paralysis resulting in total deafness in one ear and mixed deafness in the other ear

ZUSAMMENFASSUNG

Viralinfektion und Re-infektion durch den Mumpsvirus ist wahrscheinlich eine wichtige Ursache plotzlicher perceptions Taubheit Der durch diese Ansteckung verursachte Schade mag sich auf die Cochlea beschränken aber auch das Gehirn mit einbeziehen. Ebenfalls kann eine heftige nicht bacterielle Entzündung des ausseren Ohres, des inneren Ohres und des Mastoids auftreten.

Die Fälle zweier Patienten sind angegeben, d.h eines an einer viralen Lungenentzundung und plotzlicher Taubheit leidenden, sowie eines Patienten mit heftiger atypischer Mastoiditis, Myringitis und Facialislahmung, welche die vollige Taubheit in einem Ohr und gemischte Taubheit im anderen Ohr zur Folge hat.

REFERENCES

DISHOECK, H A. E. VAN and BIERMAN, Th. A. Surdite de perception brutale et les infections dues à des virus Rapport d'Audiologie Paris 1955

DISHOECK, H. A E. VAN and BIERMAN, Th. A. Sudden perceptive deafness and viral infection. Annals of Otol., Rhinol and Laryngology, *66* 963 (1957)

FOWLER, E P Sudden deafness Annals of Otol, Rhinol. and Laryngology *59* 980 (1950)

HALLBERG, O E Sudden deafness of obscure origin The Laryngoscope *66* 1237 1267 (1956)

SAUNDERS, W M and LIPPY, W H Sudden deafness and Bell s palsy A common cause. Annals of Otol, Rhinol. and Laryngology *68* 830 (1959)

SCHUKNECHT, H F Clinical Trends in Otolaryngology New York. Vol. 1, no 2 July 1962.

ted to our clinic because of this bilateral deafness, severe earache needing morphia, bilateral otitis externa and right sided facial paralysis By exploration of both mastoids it was found that they were filled with whitish tissue and a watery brownish fluid Histology chronic inflammation with round cell infiltration and foreign body reaction resembling in some parts granulation tissue Bact Cultures from mastoid and external meatus negative t b neg, moulds neg, virus cultures as far as done neg Temp 39°, R C S R 40/64 White cells /mm³ 8000 — leuco 75% Lymph and Mono 25%

Audiogram R total deafness, L perceptive deafness of 50dB

January 4th 1962 general condition much improved R C S R 2/5 Temp normal. R ear total deafness L ear air conduction 50dB, bone conduction 30dB hearing loss Facialis R. total paralysis February 5th facialis decompression followed by prompt recovery March April R ear total deafness No vestibular reactions L ear air conduction 50dB, bone conduction 30dB hearing loss The patient has been rehabilitated with a hearing aid

Epicrisis Otitis externa media, mastoiditis and facial neuritis without any bacterial infection Antibiotics without any effect Atypical lymphocytic mastoid infiltration R ear total perceptive deafness L ear recovery of a perceptive deafness but remaining of transmission deafness probably caused by adhesions Presumably this atypical ear inflammation is caused by an undetected virus

Discussion Myringitis as a complication of viral infection e.g. in influenza is wellknown The same holds true for benign lymphocytic meningitis and encephalitis Damage done by viral infection to the middle ear is not familiar to the otologist



Microphotograph shows ear of a patient who experienced sudden deafness Right ear was normal, left ear diseased Note shrinkage of tectorial membrane organ of Corti and stria vascularis There was also a total loss of hair cells in saccule (By courtesy of Dr Schuknecht)

Recently Schuknecht c s examined the temporal bones of four patients with unilateral sudden deafness of unknown etiology According to him the changes are similar to those which occur in human labyrinthitis of known viral etiology and are unlike the pathological changes resulting from experimental vascular occlusion in animals He also notes as we do that the evidence suggests that mumps or a related virus may be an important etiological factor in sudden perceptive deafness

SUMMARY

Viral infection and reinfection with the mumps virus is probably an important cause of sudden perceptive deafness The damage done by this infection may be restricted to the cochlea but may also involve the brain and may also cause severe sterile inflammation of the external ear inner ear and mastoid Two patients are reported one suffering from viral pneumonia and sudden deafness and the other from severe atypical mastoiditis myringitis and facial paralysis resulting in total deafness in one ear and mixed deafness in the other ear

centers as the increase in actionpotential frequency produced by the ampullopetal *endolymph movement in the horizontal canal*

Further studies of the electronystagmographic response in the neck muscles of pigeons using simple slicks indicated that the transmission of an ampullopetal stimulation of the horizontal canal followed a monosynaptic pathway to the muscle, giving a response after 2,5—3 millisec

Rapidly repeated clicks or a tone produced two changes in this response
1 a sequence of increases and decreases of the monosynaptically induced muscle potentials, and 2 a long series of afterdischarges

The action of reverberating and reexciting internuncial neurons in the nuclei and reticular formation seems undoubtedly to give the best support for an explanation of these phenomena and thereby the waxing and warning of the nystagmus movements recorded from the pigeons neck muscles

ACKNOWLEDGEMENT

To dr N G HENRIKSSON and dr L GLEISNER and the ENT-department in Lund Sweden, we wish to express our gratitude for the help granted for some of the experiments.

GRUNDSÄTZLICHES ZUR ATMUNG ALS RHINOLOGISCH KIEFERORTHOPÄDISCHES GRENZPROBLEM

von

A ECKERT MÖBIUS
Halle/S Deutschland

Umstrittene rhinologisch kieferorthopadische Grenzprobleme waren der Anlaß zur Überprufung der bisher keineswegs einheitlich beantworteten Frage, welche Krafte bei der Aufrechterhaltung der fur die Nasenatmung erforderlichen Mund abdichtung wirksam sind und welche weiteren funktionellen Folgen sich bei Nasen bzw Mundatmung daraus ergeben

Der Zahnarzt Mezger[1] hatte schon im Jahre 1875 aus sehr aufschlußreichen Selbst beobachtungen gefolgert, daß der *luftdichte Abschluß der Mundhohle* nach Schließen des Mundes und dem automatisch nachfolgenden Verschlucken des angesammelten Speichels wahrend der weiteren Mundruhe durch die am Gaumen haftenbleibende *Zunge* erfolgt und das diese samt dem Unterkiefer und den angrenzenden Weichteilen vom außeren *Luftdruck* getragen wird

Der Physiologe Donders[1] der diesen Folgerungen zustimmte veroffentlichte dazu eine Nachschrift in der er auf Grund eigener manometrischer Untersuchungen einen umschriebenen *Saugraum* uber der Zungenmitte mit einem zwischen 2 und 4 mm Hg schwankenden negativen Druck feststellte und folgerte daraus zusatzlich daß die Zunge bei Nasenatmung durch *eigene Muskeltatigkeit* mehr oder weniger gegen den Gaumen angesogen liegt

Der innere Widerspruch, der — bei einem Dauerzustand subjectiv vollig ent spannter Mundruhe[1] — in dieser *Kombination von passiver Luftdruck* und *aktiver Saug und Druckwirkung der Zunge* liegt, hat durch die Autoritat von Donders zunehmende Verbreitung in Lehre und Forschung gefunden und zahl reiche klinische Fehlschlusse gezeitigt, die auch heute noch nicht restlos beseitigt sind

Eigene in der Deutchen Zahn Mund- und Kieferheilkunde Bd 18 (1953) veroffentlichte Untersuchungen bestatigten die subjektiven Beobachtungen Mezgers und brachten durch die *objektive Darstellung des Verhaltens der Zunge* unter normalen und pathologischen Verhalt nissen an Hand von *Rontgenkontrast Profilaufnahmen* des Gesichtsschadels eine eindeutige Klarung der ursachlichen Zusammenhange Nach Mundschluß und automatisch nachfolgendem Schluckakt liegt die Zunge bei ungestorter Nasenatmung in ganzer Ausdehnung luftdicht den Kiefern und dem harten und weichen Gaumen an Es entsteht *subjektiv* ein Zustand vollig *muskelspannungsfreier Mundruhe* bei dem *objektiv* — im Rontgenbild — außer einem geringen Auseinanderweichen der Zahnreihen ein schmaler spitz-ovalarer *Spaltraum* uber der Mitte der Zunge sichtbar wird (Abb 1) Bei tiefer Inspiration oder bei manuellem Abwartsdrangen des Kehlkopfes vergroßert sich der Spaltraum und last dadurch seine uberwiegende Abhangigkeit von respiratorischen *elastischen Zugkraften des Tracheobronchialbaumes* erkennen die auch bei der Durchtrennung der Trachea in dem Auseinanderweichen der Schnittenden deutlich sichtbar werden

Aus diesen mit den subjektiven Beobachtungen vollig ubereinstimmenden ob jektiven Befunden ergibt sich daß der luftdichte Abschluß der Mundhohle bei Mundruhe nicht muskular sondern *ausschließlich durch den atmospharischen Druck*

[1]) Pflugers Arch 10 (1875)

auf die dem annähernd halbkugeligen Hohlraum der Mundhöhle allseits luftdicht angelagerte Zunge — in Verbindung mit der Adhäsionswirkung der aneinander gelagerten Weichteile — aufrecht erhalten wird

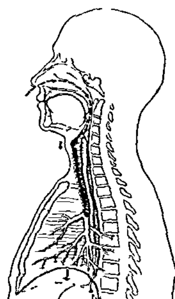


Abb 1 Paramedianer Sagittalschnitt mit Einzeichnung des spaltförmigen Unterdruckraumes über der Zunge und der bei Nasenatmung wirksamen elastischen Zugkräfte des Tracheobronchialbaumes (Pfeile ↑)

Der sogenannte "Saugraum" über der Zungenmitte erweist sich also in seiner Verknüpfung mit einer ursächlichen aktiven Betätigung der Zunge als eine *irreführende Fehlbezeichnung* Er muß eindeutiger als *Unterdruckraum* bezeichnet werden, denn er ist ausschließlich das *Ergebnis der insgesamt etwas stärkeren Zugkräfte, die dem Luftdruck entgegen wirken* Diese bestehen teils aus den je nach der Körperhaltung wechselnden Schwerkraftauswirkungen der Zunge, des Unterkiefers und der angrenzenden Weichteile, hauptsächlich aber aus den dauernd wirksamen elastischen Zugkräften des durch Bänder fest mit der Zunge verbundenen Tracheobronchialbaumes

Bei *normaler Nasenatmung* besteht also während der Mundruhe in der durch die Zunge abgedichteten Mundhöhle stets ein geringer *Unterdruck*

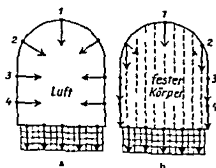


Abb 2 Physikalische Auswirkungen von Zugkräften in Richtung und Länge der senkrechten basalen Pfeile auf die Wände eines verlängert halbkugeligen festen Hohlkörpers.
a) Wenn dieser mit Luft oder Flüssigkeit, b) wenn dieser mit einem festen Körper ausgefüllt ist.

Nach den in Abb. 2 dargestellten physikalischen Gesetzmäßigkeiten wirkt er sich, da die Zunge mehr einem festen als einem flüssigen Aggregatzustand entspricht, am harten Gaumen als dem Gegenpol der überwiegenden elastischen Zugkräfte am stärksten aus und führt hier durch teilweise Lösung der Adhäsion zur Ausbildung einer *Unterdruckraumes*. An den vorderen und seitlichen Begrenzungen der Mundhöle dagegen ist er wesentlich geringer und wird nur bei verstärkter Inspiration und der damit verbundenen stärkeren Zugwirkung auf die Zunge auch subjektiv spürbar.

Aus diesen biologisch-physikalischen Gesetzmäßigkeiten ergeben sich folgende klinische Folgerungen:

1. Alle früheren Auffassungen, die sich auf eine *aktive muskuläre Tätigkeit der Zunge* bei normaler Atmung und eine dadurch bedingte *Druckwirkung* auf die Kiefer stützen, sind hinfällig.
2. Die von Loebell vertretene Möglichkeit *kieferkompressorischer Auswirkungen einer erschwerten Nasenatmung* infolge dadurch verstärkter Druckdifferenzen und des Überwiegens der negativen inspiratorischen Druckkomponente ist gleichfalls nicht haltbar. Die dabei verstärkte inspiratorische Zugbelastung der Zunge würde im Gegenteil eher eine Abflachung des Gaumens begünstigen.
3. Auch die von Kantorowicz und Korkhaus bei *erschwerter Nasenatmung* beschriebene Wechselatmung (Einatmung durch die Nase, Ausatmung durch den Mund), die durch den verstärkten negativen Inspirationsdruck auf die dem Gaumen anliegende Zunge eine *Kieferkompression* begünstigen soll, mußte, wenn diese Voraussetzungen zuträfen, zu einem entgegengesetzten Ergebnis führen. In Wirklichkeit kann aber bei einem derartig raschen Dauerwechsel zwischen Öffnung und Schließung des Mundes eine Anlagerung der Zunge an den Gaumen überhaupt nicht erfolgen.
4. Die im Endergebnis sich ausgleichenden geringen Druckdifferenzen der flachen *Mundatmung* sind als *direkt* formende Kräfte abzulehnen.
5. *Indirekt* aber kann sich nach den hier mitgeteilten Ergebnissen eine lang anhaltende Mundatmung im Kindesalter durch die *Ausschaltung der bei Nasenatmung* wirksamen elastischen Zugkräfte auf die dem Gaumen luftdicht anliegende Zunge durchaus im Sinne einer *verminderten Abflachung des Gaumens* auswirken.

Gemeinschaftsuntersuchungen an 1300 rhinologisch und zahnärztlich sorgfältig begutachteten sechsjährigen Kindern ergaben ein Überwiegen des hohen Gaumens bei Kindern mit behinderter Nasenatmung und umgekehrt des flachen Gaumens bei normal atmenden Kindern um je ein Drittel bei gleichmäßiger Verteilung des mittelhohen Gaumens auf beide Atmungstypen.

Trotz der weitgehend anlagebedingten Entwicklung der hohen Gaumen spricht dieses Ergebnis für eine die *Gau[illegible]flachung begünstigende Auswirkung der normalen Nasenatmung* und unterst[illegible] Forderung nach einer möglichst frühzeitigen Beseitigung aller Ursachen [illegible] Nasenatmung beeinträchtigen.

LA BIOPSIE LARYNGÉE EST-ELLE TOUJOURS INDIQUÉE?

par

F G EEMAN M Sc

Gand Belgique

La pathologie laryngee pose parfois des problemes de diagnostic fort delicats et c est cependant d un diagnostic exact que decoule la therapeutique adequate La sanction en sera souvent la guérison du malade tandis qu une erreur de diagnostic ou une rectification trop tardive peut signifier un arret de mort Les affections du *larynx* d aspect *tumoral* *constituent un exemple typique du problème,* parfois délicat qui peut se poser au laryngologue

Un diagnostic indiscutable est il toujours urgent? Quels moyens est-on en droit d appliquer pour y parvenir et quand doit-on y recourir?

En principe dans toute lesion hypertrophique laryngee une biopsie est indiquee *et devrait etre appliquee* L examen histologique d un fragment préleve correctement a le plus de chance de nous mettre à l abri d une erreur de diagnostic et de nous dicter la therapeutique adéquate et efficace

Les biopsies laryngees indiscutablement necessaires sont fréquemment pratiquees trop tardivement Les laryngologues sont unanimes a regretter la negligence prolongee des interesses qui acceptent durant de longs mois une raucité progressive mais indolore et ne viennent les consulter que lorsqu une dyspnee progressive ou de la dysphagie viennent s y surajouter L image laryngee decouverte en ce moment est presque toujours desolante et par sentiment d humanité le specialiste epargnera les reproches steriles a cet homme qu il aurait pu sauver quelques mois plus tot et qui se presente a lui porteur du sceau d une echeance fatale et irrémediable

Mais ce n est pas toujours l interesse qui portera la responsabilite d un drame devenu inevitable mais parfois l optimisme injustifie du médecin de famille que la raucite de son malade n alerte pas et qui perd des semaines ou des mois en essais therapeutiques steriles

Le laryngologue lui meme n est pas toujours à l abri de reproches soit qu un examen trop peu minutieux ne lui a pas fait découvrir la petite tumeur de la commissure antérieure ou de la base de l epiglotte soit qu au cours d une consultation chargée il ait negligé de pratiquer chez son malade a reflexes violents l anesthesie de surface qui lui aurait permis d avoir une vision complète et méthodique de tout l entonnoir larynge.

Si la necessite impérieuse d une biopsie precoce dans toute lésion laryngee suspecte de malignite ne sera contestee par personne cette regle doit-elle toujours etre appliquee avec son absolutisme apparent?

Les principes médicaux s appliquant à des organismes biologiques ne peuvent

Nach den in Abb. 2 dargestellten physikalischen Gesetzmäßigkeiten wirkt er sich, da die Zunge mehr einem festen als einem flüssigen Aggregatzustand entspricht, am harten Gaumen als dem Gegenpol der überwiegenden elastischen Zugkräfte am stärksten aus und führt hier durch teilweise Lösung der Adhäsion zur Ausbildung eines *Unterdruckraumes*. An den vorderen und seitlichen Begrenzungen der Mundhöhle dagegen ist er wesentlich geringer und wird nur bei verstärkter Inspiration und der damit verbundenen stärkeren Zugwirkung auf die Zunge auch subjektiv spürbar.

Aus diesen biologisch-physikalischen Gesetzmäßigkeiten ergeben sich folgende klinische Folgerungen:

1. Alle früheren Auffassungen, die sich auf eine *aktive muskuläre Tätigkeit der Zunge* bei normaler Atmung und eine dadurch bedingte *Druckwirkung* auf die Kiefer stützen, sind hinfällig.

2. Die von Loebell vertretene Möglichkeit *kieferkompressorischer Auswirkungen einer erschwerten Nasenatmung* infolge dadurch verstärkter Druckdifferenzen und des Überwiegens der negativen inspiratorischen Druckkomponente ist gleichfalls nicht haltbar. Die dabei verstärkte inspiratorische Zugbelastung der Zunge würde im Gegenteil eher eine Abflachung des Gaumens begünstigen.

3. Auch die von Kantorowicz und Korkhaus bei *erschwerter Nasenatmung* beschriebene Wechselatmung (Einatmung durch die Nase, Ausatmung durch den Mund), die durch den verstärkten negativen Inspirationsdruck auf die dem Gaumen anliegende Zunge eine *Kieferkompression* begünstigen soll, müßte, wenn diese Voraussetzungen zuträfen, zu einem entgegengesetzten Ergebnis führen. In Wirklichkeit kann aber bei einem derartig raschen Dauerwechsel zwischen Öffnung und Schließung des Mundes eine Anlagerung der Zunge an den Gaumen überhaupt nicht erfolgen.

4. Die im Endergebnis sich ausgleichenden geringen Druckdifferenzen der flachen *Mundatmung* sind als *direkt* formende Kräfte abzulehnen.

5. *Indirekt* aber kann sich nach den hier mitgeteilten Ergebnissen eine lang anhaltende Mundatmung im Kindesalter durch die *Ausschaltung der bei Nasenatmung* wirksamen elastischen Zugkräfte auf die dem Gaumen luftdicht anliegende Zunge durchaus im Sinne einer *verminderten Abflachung des Gaumens* auswirken.

Gemeinschaftsuntersuchungen an 1300 rhinologisch und zahnärztlich sorgfältig begutachteten sechsjährigen Kindern ergaben ein Überwiegen des hohen Gaumens bei Kindern mit behinderter Nasenatmung und umgekehrt des flachen Gaumens bei normal atmenden Kindern um je ein Drittel bei gleichmäßiger Verteilung des mittelhohen Gaumens auf beide Atmungstypen.

Trotz der weitgehend anlagebedingten Entwicklung der hohen Gaumen spricht dieses Ergebnis für eine die *Gaumenabflachung begünstigende Auswirkung der normalen Nasenatmung* und unterstreicht die Forderung nach einer möglichst frühzeitigen Beseitigung aller Ursachen, die die Nasenatmung beeinträchtigen.

LA BIOPSIE LARYNGÉE EST ELLE TOUJOURS INDIQUÉE?

par

F G EEMAN, M Sc
Gand Belgique

La pathologie laryngee pose parfois des problemes de diagnostic fort délicats et c est cependant d un diagnostic exact que decoule la therapeutique adéquate La sanction en sera souvent la guerison du malade tandis qu'une erreur de diagnostic ou une rectification trop tardive peut signifier un arret de mort Les affections du larynx, d aspect tumoral, constituent un exemple typique du problème, parfois delicat, qui peut se poser au laryngologue

Un diagnostic indiscutable est il toujours urgent? Quels moyens est-on en droit d appliquer pour y parvenir et quand doit-on y recourir?

En principe, dans toute lesion hypertrophique laryngee, une biopsie est indiquée et devrait etre appliquee L'examen histologique d un fragment, prélevé correctement, a le plus de chance de nous mettre a l'abri d une erreur de diagnostic et de nous dicter la therapeutique adequate et efficace

Les biopsies laryngees, indiscutablement necessaires, sont fréquemment pratiquees trop tardivement Les laryngologues sont unanimes à regretter la négligence prolongee des interesses qui acceptent durant de longs mois une raucité progressive mais indolore et ne viennent les consulter que lorsqu'une dyspnée progressive ou de la dysphagie viennent s'y surajouter L image laryngée découverte en ce moment, est presque toujours desolante et, par sentiment d humanité, le spécialiste épargnera les reproches steriles à cet homme qu'il aurait pu sauver quelques mois plus tot et qui se presente a lui, porteur du sceau d une échéance fatale et irrémediable

Mais ce n'est pas toujours l interesse qui portera la responsabilité d'un drame devenu inevitable mais parfois l optimisme injustifie du médecin de famille que la raucite de son malade n alette pas et qui perd des semaines ou des mois en essais therapeutiques stériles

Le laryngologue lui meme n est pas toujours à l abri de reproches, soit qu'un examen trop peu minutieux ne lui a pas fait découvrir la petite tumeur de la commissure antérieure ou de la base de l epiglotte, soit qu au cours d'une consultation chargee il ait negligé de pratiquer chez son malade à reflexes violents l anesthésie de surface qui lui aurait permis d avoir une vision complète et méthodique de tout l entonnoir laryngé

Si la necessite imperieuse d une biopsie precoce dans toute lésion laryngée suspecte de malignite ne sera contestee par personne, cette regle doit-elle toujours etre appliquée avec son absolutisme apparent?

Les principes médicaux s appliquant a des organismes biologiques ne peuvent

avoir la rigidité de formules mathématiques et pour répondre à cette première question il convient d'en poser une autre qui en conditionne la solution

La biopsie est-elle totalement inoffensive et absente de tout risque? Si tel n'est pas le cas, la réponse à la première question doit devenir plus nuancée et l'indication de la biopsie doit être minutieusement soupesée pour en découvrir une contre-indication éventuelle

Or, personne ne niera qu'en ouvrant des voies lymphatiques et sanguines et en mobilisant des cellules néoplasiques, la biopsie peut déclencher la propagation de celles-ci au voisinage sous forme de métastases ganglionnaires ou leur essaimage à distance Il en découle que, si le principe de la biopsie laryngée ne peut se discuter, sa mise en pratique ne peut être envisagée que s'il y a plus d'avantages probables à en retirer que de risques inutiles à faire encourir

Or, dans certains cas, la biopsie n'est pas urgente et son exécution peut être retardée de quelques semaines pour devenir parfois inutile à la suite d'un traitement strictement médical comportant essentiellement la mise au repos complet du larynx la suppression totale du tabac et une médication décongestionnante d'effet d'ailleurs plus psychologique que réel Cette laryngite chronique banale, d'aspect cependant inquiétant, ne justifiait pas, même chez ce gros fumeur, une biopsie immédiate La régression rapide des phénomènes inflammatoires éclaircira le diagnostic tandis que leur maintien ou leur accentuation aggravera nos soupçons et supprimera nos scrupules en l'espace de huit à quinze jours Dans d'autres cas le traitement d'épreuve de silence absolu diminuera un oedème surajouté et laissera rapidement découvrir une lésion tumorale suspecte qui ne sera plus noyée par des réactions de voisinage et pour laquelle, cette fois, l'opportunité d'un contrôle biopsique ne se discute plus

L'abstention momentanée d'une biopsie cependant défendable est plus indiquée encore en cas de cordite unilatérale dont on ne peut écarter a priori une origine tuberculeuse La biopsie aura écarté la malignité et prouvé l'étiologie infectieuse spécifique mais aura transformé une lésion fermée rapidement curable par les tuberculostatiques en une lésion ouverte susceptible de s'ulcérer, de se surinfecter et de guérison plus aléatoire

Une courte épreuve thérapeutique par tuberculostatique précédant une biopsie ultérieure éventuelle aura dans les cas douteux une valeur diagnostique incontestable par la positivité ou négativité de son résultat et cela au prix d'inconvénients ou de risques qu'on peut considérer comme mineurs Dans des cas similaires l'abstention passagère d'une biopsie prématurée est parfaitement justifiable et le principe de ce qu'on peut dénommer la biopsie différée n'est pas erroné

L'âge avancé du sujet porteur d'une lésion suspecte peut également constituer une contre-indication à une biopsie que l'aspect de la tumeur semblerait cependant exiger La lenteur de l'évolution des tumeurs malignes chez les vieillards dont statistiquement les chances de survie s'amenuisent de jour en jour doit nous faire hésiter à fouetter par notre biopsie une lésion assez quiescente qui ne sera probablement pas responsable de la mort si nous la laissons évoluer à son rythme ralenti

Ici se pose donc un problème d'opportunité dont de multiples élements, souvent contradictoires, doivent être soupeses dans chaque cas individuel avant de prendre la decision de pratiquer la biopsie ou de s'en abstenir

L'avenir seul precisera ulterieurement si la décision que nous avons prise avait été sage ou au contraire regrettable

Independamment de l âge du malade, *son état physique nettement compromis* peut poser le meme dilemne En excluant le cardiaque menacé de décompensation et chez lequel la biopsie elle-même constitue une manoeuvre chirurgicale susceptible de déclencher l'accident mortel et doit etre rejetée, il existe des tares physiques qui excluraient l'application des sanctions thérapeutiques qui découleraient *obligatoirement d'une biopsie positive*

Dans cette éventualite, la biopsie n'a plus aucun sens et ne pourrait etre que nefaste Il en decoule qu'un examen médical complet et minutieux peut etre nécessaire non pas „apres" mais , avant" une biopsie envisagée afin de determiner au prealable l'operabilite ou l inoperabilite ou encore la resistance à un traitement radiotherapique incontestablement débilitant

L'operabilite ayant ete reconnue, le fait que toute biopsie comporte l'eventualité d'une therapeutique consecutive active et d'application immédiate ne sera pas conteste Le malade doit en être prevenu, doit connaître l'importance du geste qu'on envisage et doit au prealable accepter de se soumettre a la decision therapeutique qui s'imposera

Non prévenu, le malade risque de se rebiffer en presence du dilemne qu'on lui *aura pose trop tard et sa decision*, qu'il n'aura pas *eu le temps de mûrir*, sera souvent negative ou dilatoire La biopsie, dans ce cas, aura ete nefaste

Elle le sera encore plus si le malade, prevenu de la decision qu'il aura à prendre, refuse d'emblee toute therapeutique ulterieure, tant radiothérapique que chirurgicale Pratiquer une biopsie dans un tel climat de negativisme constitue une erreur psychologique venant aggraver un acte chirurgical inutilement nocif par le traumatisme physique et psychique qu'il ne manquera de provoquer

Ces quelques considerations laisseront deviner qu'une *règle de conduite rigide* et incontestee souffrira parfois quelques exceptions, dictees uniquement et exclusivement par l interet du malade a l encontre parfois de notre curiosite scientifique

avoir la rigidité de formules mathématiques et pour répondre à cette première question il convient d'en poser une autre qui en conditionne la solution

La biopsie est elle totalement inoffensive et absente de tout risque? Si tel n'est pas le cas, la réponse à la première question doit devenir plus nuancée et l indication de la biopsie doit être minutieusement soupesée pour en découvrir une contre indication éventuelle

Or, personne ne niera qu'en ouvrant des voies lymphatiques et sanguines et en mobilisant des cellules neoplasiques, la biopsie peut déclencher la propagation de celles-ci au voisinage sous forme de metastases ganglionnaires ou leur essaimage à distance Il en découle que, si le principe de la biopsie laryngée ne peut se discuter, sa mise en pratique ne peut être envisagée que s'il y a plus d'avantages probables à en retirer que de risques inutiles à faire encourir

Or, dans certains cas, la biopsie n'est pas urgente et son exécution peut être retardee de quelques semaines pour devenir parfois inutile à la suite d un traitement strictement médical, comportant essentiellement la mise au repos complet du larynx, la suppression totale du tabac et une médication decongestionnante d'effet d'ailleurs plus psychologique que réel Cette laryngite chronique banale, d'aspect cependant inquietant, ne justifiait pas, même chez ce gros fumeur, une biopsie immediate La regression rapide des phénomenes inflammatoires eclaircira le diagnostic tandis que leur maintien ou leur accentuation aggravera nos soupçons et supprimera nos scrupules en l'espace de huit à quinze jours Dans d'autres cas le traitement d'épreuve de silence absolu diminuera un oedème surajouté et laissera rapidement découvrir une lésion tumorale suspecte qui ne sera plus noyée par des réactions de voisinage et pour laquelle, cette fois, l'opportunité d'un controle biopsique ne se discute plus

L'abstention momentanée d'une biopsie cependant defendable est plus indiquée encore en cas de cordite unilatérale dont on ne peut écarter à priori une origine tuberculeuse La biopsie aura écarté la malignité et prouvé l'étiologie infectieuse spécifique mais aura transformé une lesion fermee rapidement curable par les tuberculostatiques en une lesion ouverte susceptible de s'ulcérer, de se surinfecter et de guersion plus aleatoire

Une courte épreuve thérapeutique par tuberculostatique, précédant une biopsie ulterieure éventuelle, aura dans les cas douteux une valeur diagnostique incontestable par la positivite ou negativite de son resultat et cela au prix d'inconvénients ou de risques qu'on peut considérer comme mineurs Dans des cas similaires, l'abstention passagère d'une biopsie prematurée est parfaitement justifiable et le principe de ce qu on peut denommer la biopsie differee ' n'est pas erroné

L'age avancé du sujet porteur d'une lésion suspecte peut également constituer une contre indication a une biopsie que l'aspect de la tumeur semblerait cependant exiger La lenteur de l evolution des tumeurs malignes chez les vieillards dont statistiquement les chances de survie s'amenuisent de jour en jour, doit nous faire hésiter a fouetter par notre biopsie une lésion assez quiescente qui ne sera probablement pas responsable de la mort si nous la laissons évoluer à son rythme ralenti

intervention been omitted While it must be admitted that statistical percentages are a valuable aid, the determination of an operative risk is a complicated process This risk, after all, is not solely dependent on the surgeon Factors of undeniable importance in this respect include the general standard of the Hospital or Sanatorium, the type of operation performed, the skill of the anaesthetist Last but not least, factors supplied by the patients must be taken into account, these include age, *nutritional condition, various accompanying affections,* and particularly the psychological condition

TABLE I

Jaar	*Extrapleur* pneumothorax	Prim †	Thoraco-plasty	Prim. †	Resection	Prim. †
1938 t/m 1947	33		150		11	1
1948	54		58		17	—
1949	16		33		78	4
1950	15		19		143	6
1951	2		21		212	2
1952	—		18		198	3
1953	—		20		186	3
1954	1		19		175	4
1955	—		16		140	3
1956	—		11		134	—
1957	—		4		129	1
1958	—		4		150	2
1959	—		2		126	—
1960	—		6		82	—
1961	—		4		82	—
1962 tot 1 7	—		—		38	—
	121	4 †	385	21 †	1901	29 †
		3 3%		5 4%		1 5%

In pulmonary resection, as in many other things, moderation is a commendable virtue Detailed knowledge of the anatomy of the lungs is a primary requirement, particularly for segmental resections

Much trouble can be avoided by meticulous technique, careful toilet of the bronchial stumps and closing of the pulmonary wound surface after removal of one or several segments

Finally, the choice of the time of operation within the entire constellation of therapeutic measures in pulmonary tuberculosis, largely determines the result, both in terms of complications which can be corrected, and in terms of complications so serious as to have an inevitable effect on the outcome In this respect we have learned our lesson, in the earlier days we had many more complications, sometimes fatal, than in later years when our indications were determined with greater strictness For example, we learned that a pulmonary resection is definitely to be avoided as long as the process is still active Unless this rule is adhered to, there will be complications such as bronchial fistulae, empyema, spread of the process and relapse, sometimes with fatal results

After these introductory remarks, we present a description of our material

During the period 1938—1962 (July 1st), the abovementioned 1901 pulmonary resections were performed, tuberculosis was the most frequent indication, 779 pulmonary resections being carried out for other indications

An analysis of therapeutic results obtained in cases of tuberculosis over a considerable number of years, will inevitably reveal gradual changes in surgical indications, operative techniques, and particularly in pre and postoperative treatment

Naturally, there will always be indications for pulmonary resection, e g in bronchial stenosis, and particularly in the case of relapse following conservative therapy A patient's pulmonary tuberculosis usually presents a mixture of reversible and irreversible pathological processes The improvement seen on X rays after medication, as a rule concerns the reversible changes Whenever there is an important irreversible component such as a caseous focus or cavitation, the patient is usually a prospect for surgical treatment, particularly if these changes are localized in parts of the lungs where phthisis can be expected (apicodorsal segment of the upper, and apical segment of the lower lobes)

Emphasizing the good results of resection therapy, we must bear in mind that thoracoplasty, too, has had the benefit of antituberculotic drugs since 1949 In addition it must be admitted that the loss of pulmonary function after thoracoplasty has been reduced since physiotherapy and rehabilitation of patients have received more attention

This is very apparent in my series The operative mortality of thoracoplasty diminished from 8 % prior to 1948 (12 † in 150 cases) to 3 8 % after 1948 (9 † in 235 cases) The ultimate results however, remained inferior to those of pulmonary resections A few surgeons have obtained very fine results, e g H SELLORS who had an early postoperative mortality of 2 7 % and a late postoperative mortality of 8 7 % in his thoracoplasty patients the sputum remaining positive in 16 % Yet another factor became involved To an increasing extent the choice of operation was being made with a view to pulmonary function In this respect too pulmonary resection compares favourably with collapse therapy particularly in the case of segmental resections

Anyone who wishes to champion pulmonary resection as the operation of ice, must first demonstrate that the mortality inherent to the intervention stitutes no objection to its use

Table II provides an explicit answer to this question The total operative mor ty (up to 2 months after operation) proved to be 1 5 % (29 † in 1901 ections) For segmental resections, the operative mortality was as low as 0 25 % † in 1190 segmental resections) — a percentage of the type encountered in pendectomy series!

TABLE II

Period 43—1/7 62	Pneum 264	Prim † 21 (7 9%)	Late † 33
Resections for lung tuberculosis	Lob 403	Prim † 4	Late † 12
	Lob + S R 44	Prim † 1	Late † —
	S R. 1190	Prim † 3 (0 25%)	Late † 3
Total resections	1901	Prim † 29 (1.5%)	Late † 48

The primary mortality for all lobectomies, segmental resections and combinations f these two, was 0 48 % (8 † in 1637 resections)

Pneumonectomy — even today an operation which places great stress on the atient — had a much higher mortality 7 9 % (21 † in 264 pneumonectomies) This is not surprising because this series includes the many usually very difficult neumonectomies performed for destroyed lung, particularly in the early years ince they have been eliminated, our material has also shown a considerable decrease n pneumonectomies in favour of lobectomies, and particularly in favour of seg mental resections, in the course of the years (cf Table III)

The causes of death within two months of the operation are presented in

Table III, while Table IV concerns the 48 late deaths There was 1 death during the period 1943—1947, 40 deaths occured during 1948—1952, and 7 patients died during the period 1953—1962 (July 1st)

During the period 1948 through 1952, when indications were less strict at our Centre, while the duration of pre and postoperative management was in fact too short, the results of resection therapy were poorer than those obtained since

Among the 648 resections of the firstmentioned period there were 15 primary deaths (2 3 %) and 41 late deaths

TABLE III

PRIMARY MORTALITY PER 1/7 1962

(within 2 months postoperatively)

Causes of death	Total	Pneum	Lob	Lob + S R	S R
Thrombosis emboly	8	1	4	1	2
Acute cardiac failure (shock)	10	9	—	—	1
Anoxia (anaesth edema glottis)	4	4	—	—	—
Atelectasis (contralateral)	1	1	—	—	—
Empyema + progressive disease	2	2	—	—	—
Pneumothorax (contralateral)	1	1	—	—	—
Rupture sup caval vein	1	1	—	—	—
Tracheomalacy	1	1	—	—	—
Cause unknown	1	1	—	—	—
Total	29 †	21 †	4 †	1 †	3 †

Among the 1242 resections since performed, there were 13 primary deaths (1 %) and only 7 late deahts

Although we may fortunately claim a very low mortality of resection therapy in our estimate of the operative risk we must not be blind to the complications seen both serious and less serious The latter category includes delayed expansion of remaining lung tissue In nearly all cases respiratory exercises led to a good result In some cases bronchoscopy was resorted to for suction drainage of secretions from the bronchial tree A bronchial toilet of this type was nearly always followed by satisfactory expansion

The number of wound infections remained within reasonable limits They were not alarming considering the size of the wound made in a thoracotomy

In 5 patients a secondary thoracotomy was necessitated by a severe postoperative haemorrhage All patients recovered

The main factors unfavourably influencing primary and late operative risks are

bronchial fistulae, with empyema formation, and relapses, with reactivation and dissemination of the tuberculous lung process Fortunately, the majority of these patients were cured by conservative measures and partly by supplemental operations such as secondary resection (78) or thoracoplasty In the past few years, the number of relapses following resection therapy has significantly decreased

TABLE IV

LATE MORTALITY PER 1/7 1962

Causes of death	Total	Pneum	Lob	S R
Bronchial fistula empyema spread of disease	14	7	7	—
Bronchial fistula, empyema, amyloidosis	2	2	—	—
Aspec pneum croup pneum virus pneum	8	7	1	—
Aspiration pneum (oesoph fistula)	1	1	—	—
Meningitus tuberculosa	1	—	1	—
Apoplexia	1	1	—	—
Peritonitis diffusa (appendicitis)	1	1	—	—
Murdered by husband	1	1	—	—
Suicidum	1	1	—	—
Carcinoma uteri	1	—	—	1
Pericarditis constrictiva	2	2	—	—
Lungcancer (oatcell)	1	—	—	1
Fusion operation Albee (spondylitis)	1	—	1	—
Carcinosis peritonei	1	1	—	—
Mesenterial emboly	1	—	—	1
Nephrosis	1	1	—	—
Heart failure cor pulmonale	10	8	2	—
Total	48 †	33 †	12 †	3 †

Non–t b cause of death 31†

In a previous publication on the fates of 637 patients followed up over more than 5 postoperative years it was reported that the sputum had become negative in 98 1 % while 93 8 % had completely resumed work

QUARRY TECHNIQUE FOR EXTIRPATION OF THE EXTERNAL AUDITORY CANAL

by

EDMUND P FOWLER Jr *
New York N Y U S A.

For many years when a malignant growth was found in the external auditory canal it has been the custom of surgeons to remove the canal and its environs piecemeal thus often injuring the facial nerve Some five years ago when confronted by a patient whose external auditory canal showed a squamous cell epithelioma but normal hearing indicating that the invasion had not yet entered the middle ear the author after minimal experimentation on autopsy material evolved the following method for removing the external auditory canal en bloc without injury to the facial nerve

The mastoid was entered as for a simple mastoidectomy, i e the posterior canal wall was left intact while removing the mastoid air cells and the ossicles were exposed by burring away the bone in the attic wall into the zygomatic region The facial nerve was decompressed from the stylomastoid foramen to the geniculate ganglion and lifted from its bed To do this it was necessary to remove the incus and the head of the malleus very much as is done in the Sourdille fenestration operation With a fine perforating burr drill holes are then made through the floor of the open Fallopian canal directing the drill holes into the middle ear Each drill hole was about two millimeters apart In the zygomatic area the bone was burred down directly to the back of the cartilage of the temporomandibular joint

A wide circular incision was now made in the concha of the auricle so as to include a wide margin of normal skin and tissue beyond the cancerous growth The incision was extended well under the external auditory canal to obtain access to the most difficult portion of the operation which is the anterior sulcus of the external auditory canal As originally performed on the first patient the perforating drill was inserted so as to make a series of holes in the middle of the mass of bone below the external canal Subsequently Dr Conley and Dr Novack [1] who became intrigued with this technique omitted this procedure and simply dissected the soft tissue including the facial nerve away from the inferior surface of the temporal bone This is somewhat more dangerous for stretching the facial nerve but it gives a wider margin of tissue and makes the removal of the inferior margins somewhat simpler

After the external auditory canal has been surrounded by perforation holes a medium sized sharp osteotome was placed between the holes With sharp blows

* *Columbia Presbyterian Medical Center New York N Y*

bronchial fistulae, with empyema formation, and relapses, with reactivation and dissemination of the tuberculous lung process Fortunately, the majority of these patients were cured by conservative measures, and partly by supplemental operations such as secondary resection (78) or thoracoplasty In the past few years, the number of relapses following resection therapy has significantly decreased

TABLE IV

LATE MORTALITY PER 1/7 1962

Causes of death	Total	Pneum	Lob	S R
Bronchial fistula empyema, spread of disease	14	7	7	—
Bronchial fistula empyema amyloidosis	2	2	—	—
Aspec pneum croup pneum , virus pneum	8	7	1	—
Aspiration pneum (oesoph fistula)	1	1	—	—
Meningitis tuberculosa	1	—	1	—
Apoplexia	1	1	—	—
Peritonitis diffusa (appendicitis)	1	1	—	—
Murdered by husband	1	1	—	—
Suicidum	1	1	—	—
Carcinoma uteri	1	—	—	1
Pericarditis constrictiva	2	2	—	—
Lungcancer (oatcell)	1	—	—	1
Fusion operation Albee (spondylitis)	1	—	1	—
Carcinosis peritonei	1	1	—	—
Mesenterial emboly	1	—	—	1
Nephrosis	1	1	—	—
Heart failure cor pulmonale	10	8	2	—
Total	48 †	33 †	12 †	3 †

Non–tb cause of death 31[1]

In a previous publication on the fates of 637 patients followed up over more than 5 postoperative years it was reported that the sputum had become negative in 98 1 % while 93 8 % had completely resumed work

A PROPOS DU PHÉNOMÈNE D'INTERDÉPENDANCE COCHLÉAIRE RÔLE POSSIBLE DU CORPS CALLEUX

par

MARCEL VAN EYCK

Bruxelles, Belgique

On sait que la réalité d'une interdépendance cochléaire a été objectivée grâce à la cophochirurgie Celle-ci a révélé que, chez un sourd bilatéral, l'amélioration postchirurgicale d'une oreille peut s'accompagner de l'amélioration de l'oreille non opérée

Diverses hypotheses explicatives ont été formulées au sujet de ce phénomène, à premiere vue surprenant (voir comptes rendus Soc Int Audiol) Toutefois, notre propos est de souligner l'intervention possible du corps calleux dans son mécanisme F BREMER et Coll ont, en effet, demontré la part prépondérante que prend cet organe dans le mécanisme de la synergie interhémisphérique

Déjà en 1939, CLAES avait observé chez le chat, que le corps calleux participe à la transmission des influx de l'aire *visuelle* corticale d'un hémisphère cérébral, à l'aire homologue de l'hémisphère opposé, la section du splenium du corps calleux ayant pour effet de reduire l'activité electrique spontannée des aires visuelles droite et gauche

Une telle action de dynamogénèse interhémispherique dépendant du corps calleux, existe également au niveau des aires *auditives* En effet F BREMER (1953) a apporté la preuve que, chez le chat „encéphale isolé", des influx provoqués par l'excitation électrique du cortex auditif d'un côté, sont transmis par le corps calleux au cortex auditif opposé, dès lors, les réponses de ce cortex aux stimulations auditives (clic) sont très nettement facilitees Ce pouvoir de facilitation cesse immédiatement avec le section du corps calleux

Il n'est pas sans intérêt de noter que l'anesthésie à la cocaïne de l'aire auditive d'un côté affaiblit transitoirement la réponse au clic de l'aire opposséo „comme si, normalement, dit BREMER, les deux aires auditives homologues exerçaient l'une sur l autre une dynamogenèse continue par leur échange mutuel d'influx commissuraux"

Par ailleurs, F BREMER et TERZUOLO (1956) ont montré que le corps calleux est capable aussi de transmettre les influx [illegible] d'un seul tractus auditif, vers les deux hémisph[illegible] ce [illegible] auteurs ont utilisé un stimulus qui sans aucun d[illegible] qu'[illegible] se sont servis d'un choc électrique excitant [illegible] ont observé, dans ces conditions, que cette [illegible] réponse corticale *bilatérale*, immédi[illegible]

De l'ensemble de ces [illegible] il résulte à l'évidence [illegible]

médiaire de la synergie des deux tractus auditifs En résumé, son activité est double

il suscite une dynamogenèse réciproque des aires auditives corticales,

il transmet des excitations unilatérales aux deux hémispheres, les influx provenant d'un hemisphère, ou d'un tractus auditif, exercent un remarquable effet de facilitation sur les réponses de l hémisphere opposé

Ces constatations, extrapolées à l homme, autorisent à suggérer le fait que le corps calleux constitue un des eléments intervenant dans le phenomene d'inter dépendance cochléaire, lui même supporté par l'interdépendance callosale des deux cortex auditifs Tout se passe comme si les influx nerveux provenant de l'oreille ameliorée chirurgicalement, transmis par le corps calleux aux deux cortex auditifs, exercent un pouvoir d'amplification sur les reponses du cortex auditif du côté non opéré aux stimulations sonores de l'oreille non opéreé

Pour conclure, nous croyons avoir apporté des eléments autorisant à penser que le corps calleux est susceptible de jouer un rôle dans le mécanisme intime du phenomene de l'interdependance cochléaire, phénomene objectivé cliniquement chez l'individu sourd.

BIBLIOGRAPHIE

BREMER, F Physiology of the corpus callosum, dans The Brain and Human Behavior, 1958, *36*, 424—448

BREMER, F et TERZUOLO, C. Transfert interhemispherique d informations sensorielles par le corps calleux.
J physiol. Paris, 1955, *47*, 105—107

CLAES, E Contribution a l etude physiologique de la fonction visuele, analyse oscillographique de l activité spontannee et sensorielle de l aire visuelle corticale chez le Chat non anesthesie.
Arch. internat. physiol., 1939, *48*, 181—237

Comptes-rendus du IVe Congrès de la Soc. Internat d Audiologie.
Padoue, Oct. 1958 Edit Audin, Lyon

QUARRY TECHNIQUE FOR EXTIRPATION OF THE EXTERNAL AUDITORY CANAL

by

EDMUND P FOWLER Jr *
New York, N Y U S A

For many years when a malignant growth was found in the external auditory canal, it has been the custom of surgeons to remove the canal, and its environs, piecemeal thus often injuring the facial nerve Some five years ago when confronted by a patient whose external auditory canal showed a squamous cell epithelioma but normal hearing indicating that the invasion had not yet entered the middle ear, the author after minimal experimentation on autopsy material evolved the following method for removing the external auditory canal en bloc without injury to the facial nerve

The mastoid was entered as for a simple mastoidectomy i e the posterior canal wall was left intact while removing the mastoid air cells and the ossicles were exposed by burring away the bone in the attic wall into the zygomatic region The facial nerve was decompressed from the stylomastoid foramen to the geniculate ganglion and lifted from its bed To do this it was necessary to remove the incus and the head of the malleus very much as is done in the Sourdille fenestration operation With a fine perforating burr drill holes are then made through the floor of the open Fallopian canal directing the drill holes into the middle ear Each drill hole was about two millimeters apart In the zygomatic area the bone was burred down directly to the back of the cartilage of the temporomandibular joint

A wide circular incision was now made in the concha of the auricle so as to include a wide margin of normal skin and tissue beyond the cancerous growth The incision was extended well under the external auditory canal to obtain access to the most difficult portion of the operation which is the anterior sulcus of the external auditory canal As originally performed on the first patient the perforating drill was inserted so as to make a series of holes in the middle of the mass of bone below the external canal Subsequently Dr Conley and Dr Novack [1] who became intrigued with this technique omitted this procedure and simply dissected the soft tissue including the facial nerve away from the inferior surface of the temporal bone This is somewhat more dangerous for stretching the facial nerve but it gives a wider margin of tissue and makes the removal of the inferior margins somewhat simpler

After the external auditory canal has been surrounded by perforation holes a medium sized sharp osteotome was placed between the holes With sharp blows

* *Columbia Presbyterian Medical Center New York N Y*

of a hammer the required portion of the temporal bone cracked away very much as a quarry expert serates away slabs of marble or granite The perforation holes, if properly made, force the crack to be in the weakened lines of cleavage and injuries to the vital structures such as the jugular bulb and facial nerve can be avoided

In the first case the lines of cleavage were made dangerously close to the malignant growth in its inferior anterior margin It was for this reason that this particular patient was treated with radiotherapy after the excision Even though the bare bone of the cavity was covered with split thickness skin graft in the usual way, the cancerous growth did not break down and now five years after the operation the patient is living and well, with a dry cavity and a conductive hearing loss of the type usually seen with a radical mastoidectomy At no time has there been any granulation or anything suggestive of recurrence of the disease

REFERENCES

1 CONLEY J J and NOVACK A J The surgical treatment of malignant tumors of the ear and temporal bone Arch of Oto-Laryng. *71* 635 1960

PARAMEDIANSTELLUNG DES STIMMBANDES BEI HOHER VAGUSLAHMUNG MIT GAUMENSEGEL- UND SCHLUNDSCHNÜRERLÄHMUNG

von

HERMANN FRENZEL *
Gottingen Deutschland

Es war fruher ublich, die Posticuslahmung als partielle Recurrenslahmung der kompletten Recurrenslahmung gegenuberzustellen Man wurde dann vorsichtiger und sprach von Recurrenslahmung mit Paramedianstellung und Recurrenslahmung mit Intermediarstellung der Stimmbander Die Frage nach den Zusammenhangen zwischen Sitz der peripheren Nervenschadigung und Stellung der gelahmten Stimm lippe blieb lebhaft umstritten bis durch die Durchschneidungsversuche *Hofers* und seines Mitarbeiters *Jeschek* eine uberzeugende Klarstellung dieser Zusammenhange erfolgt zu sein schien Recurrenslahmung fuhrt zur Paramedianstellung, Recurrens lahmung mit gleichzeitiger Lahmung des nervus laryngicus cranialis, eine komplette Laryngicuslahmung also, zur Intermediarstellung der gelahmten Stimmlippe

Die den Laryngologen bekannte Erfahrung, dass es Recurrenslahmungen mit Intermediarstellung der gelahmten Stimmlippe gibt, wenn durch den Sitz der Schadigung z B bei intrathorakalen Prozessen oder bei infralaryngealen Halsdurch schussen der nervus laryngicus cranialis mit voller Sicherheit unbeteiligt is, wurde von *Hofer* durch eine retrograde Einbeziehung der fur die Funktion des laryngicus cranialis zustandigen Ganglienzellen im Kerngebiet (retrograde Degeneration) ebenfalls ausreichend erklart

In dem Bestreben fur die bei der arztlichen Tatigkeit erforderliche Einordnung eines Krankheitsbildes unter dem Gesichtspunkt der Therapie eine pragmatische Systematik zu entwickeln, habe ich daher, fussend auf den Versuchen *Hofers* und *Jescheks*, seinerzeit als Eckpfeiler einer Skala der neurogenen Stimmband lahmungen die Recurrenslahmung mit Paramedianstellung der Stimmbander (sogen Posticuslahmung) der Recurrenslahmung mit Intermediarstellung der Stimmbander (komplette Laryngicuslahmung) gegenubergestellt

Gegen die Vollgultigkeit der *Hofer Jeschek* schen Schlussfolgerungen aus ihren zweifellos sehr bedeutsamen Durchschneidungsversuchen am Menschen wurden von *Krejci* bereits 195 Bedenken geaussert und zwar auf Grund der Feststellung, dass [illegible] die nach einer Jugularisunterbindung „im

* *Aus* [illegible] *Direktor Prof Dr Dr h c H Frenzel)*

Laufe der Nachbehandlung allmahlich" aufgetreten waren, das gelahmte Stimmband „4 mal in Medianstellung und 3 mal in Kadaverstellung" gefunden wurde

Krejci nimmt in allen seinen 7 Fallen eine hohe Vaguslahmung durch Bulbusthrombose an, zu der nach *Hofer* die Intermediarstellung des gelahmten Stimmbandes gehort hatte

Aber in den Befunden *Krejci's* ist weder die fur die hohe Vaguslahmung charakteristische gleichseitige Gaumensegel und Schlundschnurerlahmung erwahnt noch der von *Krejci* vermutete Sitz der Schadigung im Foramen jugulare genugend wahrscheinlich gemacht, so dass die ablehnende Entgegnung *Hofers* zunachts berechtigt erschien

Inzwischen hat *Mundnich* eine Beobachtung mitgeteilt, in der nach vollstandiger Vagusresektion bis zur Schadelbasis das Stimmband dieser Seite dauernd in der allerdings tumorbedingt schon vor dem Eingriff vorhandenen und von ihm auf eine Kontraktur nach anfanglichem Überwiegen der Adduktoren zuruckgefuhrten „starren Medianfixation' verblieb

Ich habe kurzlich bei 2 Patienten eine mit Gaumensegellahmung und Schlundschnurerlahmung der gleichen Seite verbundene hohe Vaguslahmung gesehen, bei der das gelahmte Stimmband ohne Excavation und ohne Aryknorpelvorfall in Paramedianstellung stand

Bei den beiden Kranken war die Lahmung ohne nennenwerte Stimmstorung eingetreten und bei den Untersuchungen war die Stimme unauffallig und praktisch normal, so dass angenommen werden muss, dass von Anfang an die Paramedianstellung vorhanden war

Es handelte sich im ersten Falle um einen 67 jahrigen Mann (Pat H, Priv.Nr 620208) mit Halslymphknotenmetastasen, bei deren Entfernung der Operateur den nervus vagus 2 Querfinger breit unterhalb der Carotisgabel durchschnitten und mit dem Metastasenpaket nahe der Schadelbasis abgesetzt hatte

Im zweiten Falle bestand bei einem 48 jahrigen Mann (Pat T, Priv.Nr 620726) ebenfalls ein Carcinom, das zu schweren in Rontgenschichtaufnahmen nachgewiesenen Zerstorungen der Schadelbasis im Bereich des foramen jugulare gefuhrt hatte

In beiden Fallen lagen zweifellos hohe Vaguslahmungen oberhalb des Abganges des nervus laryngicus cranialis vor

Es ware gesucht etwa annehmen zu wollen, dass zwar *die Gaumensegel und* Schlundschnureraste gelahmt aber die Fasern der nervi laryngici craniales von der Schadigung verschont geblieben waren Man muss also annehmen, dass es trotz kompletter Laryngicuslahmung zur Paramedianstellung des Stimmbandes ohne Aryknorpelvorfall und ohne Stimmbandexcavation also zur sogenannten Posticusstellung kommen kann

Es empfiehlt sich daher bei der Eingliederung der peripherneurogenen Stimmbandlahmungen im Rahmen einer pragmatischen Systematik nicht mehr von einer kompletten Laryngicuslahmung zu sprechen sondern besser unverbindlich der Abduktionslahmung die komplette Stimmbandlahmung gegenuberzustellen so dass es heisen sollte

Peripher neurogene Stimmbandlahmungen

Recurrenslahmung mit Paramedian stellung des Stimm bandes (Abduktionslahmung)	——— Skala ——— der Zwischenformen	Recurrenslahmung mit Intermediar-stellung des Stimm bandes (Komplette Stimm bandlahmung)

Als praktisch klinische Konsequenz ergibt sich aus dem Gesagten, dass man sich im Falle einer sogenannten Posticusstellung eines Stimmbandes nicht etwa darauf verlassen darf, dass dann keine hohe Vaguslahmung vorliegen kann Die in jedem Falle einer Stimmbandlahmung gleich welcher Form notwendige Prufung auch der Funktion des Gaumensegels und der Schlundschnurer, erganzt durch die Prufung der Glossopharyngicussensibilitat und der Accessoriusfunktion ergibt sich davon unabhangig sowieso aus der Tatsache, dass eine zentrale Schadigung vor liegen konnte, die je nach der Ausdehnung im Kerngebiet zu den verschieden artigsten Lahmungsbildern fuhren kann, die aber in den vorliegenden Ausfuhrun gen selbstverstandlich nicht berucksichtigt wurden

Die Frage, worauf die den Ergebnissen der *Hofer Jeschek*'schen Durchschnei dungsversuche widersprechenden Befunde der auch hier mitgeteilten Art beruhen muss vorlaufig offen bleiben

Es lasst sich bei der Fulle von Moglichkeiten, die von Bedeutung sein konnten wie etwa Varianten der sehr variablen Innervationsverhaltnisse unter Berucksich tigung der Vagoakzessoriusproblems, mechanisch sich auswirkende anatomische Varianten des Kehlkopfbaues, Muskelkontrakturen u a m vorlaufig nur dieses oder jenes vermuten, aber nicht beweisen, oder auch nur ausreichend wahrscheinlich machen.

LITERATURVERZEICHNIS

ARNOLD G E Zur Frage der *Stimmlippenstellung bei Recurrenslahmung* Mschr Ohrenheilk *92* 235 (1958)

FRENZEL H Pragmatische Systematik in der Hals Nasen-Ohrenheilkunde Munch Med Wschr 1956 972

HOFER, G und J JESCHEK s bei JESCHEK J Theorie und Klinik der Stimmbandlahmung Arch Ohr Nas Kehlk. Heilk. *162* 237 (1952)

KREJCI F Zur Genese der Stimmbandlahmung nach Jugularisligatur Mschr Ohrenheilk *85* 181 und 316 (1951)

HOFER G Stellungnahme zu Krejci
Ebenda 315

KECHT B Kritische Betrachtungen uber den Ablauf der Kehlkopflahmungen. Mschr Ohren-heilk. *92* 44 (1958)

CANCER OF THE LARYNX ASSOCIATED WITH BILATERAL LARYNGOCELE

Case Report

by

P G GERLINGS *

Utrecht, The Netherlands

One of the reasons for the increasing interest in laryngoceles is that concomitance of laryngocele and laryngeal carcinoma is not so uncommon

HUIZINGA, with his great clinical experience of over 40 years at the Groningen University E.N T Clinic, understood the importance of this association in terms of diagnosis and surgical treatment He demonstrated four cases in 1958, at a Meeting of the Netherlands Oto-Rhino-Laryngological Society.

Although LEROY SCHALL in 1944 described a case of laryngeal carcinoma with laryngocele observed in Boston as "perhaps the first report of a laryngocele found in association with cancer of the larynx", it was later established that the famous Vienna laryngologist MARSCHIK had found a secondary laryngocele in a case of laryngeal carcinoma as early as 1927 When demonstrating the laryngeal specimen at a Meeting of the "Wiener Laryngologische Gesellschaft", he pointed out that an ulcerating carcinoma was localized at the base of the laryngocele, which was filled with purulent mucus, he explained the laryngocele as a "Retentionserscheinung" resulting from occlusion of the ventricle from the laryngeal lumen

Was MARSCHIK too hasty in reaching the conclusion that occlusion of the ventriculus Morgagni by the tumour was the cause of the laryngocele? A subsequent publication by LINDSAY, LEBORGNE and others did in fact raise some doubt in this respect

In a paper entitled "Anatomical observations concerning the laryngeal appendix", EDWIN BROYLES described the results of a study of 50 laryngeal specimens, 10 of which showed an enlarged bilateral sacculus, while an enlarged unilateral sacculus was found in five The question is how large the sacculus must be to be described as a laryngocele At any rate the question remained as to whether these variations of the sinus of Morgagni are of significance, that is whether they predispose to the formation of a laryngocele or a laryngeal carcinoma It has been established with certainty that — in such an enlarged sinus of Morgagni — a carcinoma can develop which initially shows a paucity of symptoms This is pointed out in BROYLES's statement "What we wish to emphasize again is the fact that underneath the false cord in many larynges, there is an enlarged cavity which potentially may develop a hidden pathological condition"

* *From the Department of Oto-Rhino-Laryngology University of Utrecht (Director Prof Dr P G Gerlings)*

Peripher neurogene Stimmbandlahmungen

Recurrenslähmung mit Paramedian stellung des Stimm bandes (Abduktionslahmung)	——— Skala ——— der Zwischenformen	Recurrenslahmung mit Intermediar stellung des Stimm bandes (Komplette Stimm bandlahmung)

Als praktisch klinische Konsequenz ergibt sich aus dem Gesagten, dass man sich im Falle einer sogenannten Posticusstellung eines Stimmbandes nicht etwa darauf verlassen darf, dass dann keine hohe Vaguslahmung vorliegen kann Die in jedem Falle einer Stimmbandlahmung gleich welcher Form notwendige Prufung auch der Funktion des Gaumensegels und der Schlundschnurer, erganzt durch die Prufung der Glossopharyngicussensibilitat und der Accessoriusfunktion ergibt sich davon unabhangig sowieso aus der Tatsache, dass eine zentrale Schadigung vor liegen konnte, die je nach der Ausdehnung im Kerngebiet zu den verschieden artigsten *Lahmungsbildern fuhren kann*, die aber in den vorliegenden Ausfuhrun gen selbstverstandlich nicht berucksichtigt wurden

Die Frage, worauf die den Ergebnissen der *Hofer Jeschek*'schen Durchschnei dungsversuche widersprechenden Befunde der auch hier mitgeteilten Art beruhen muss vorlaufig offen bleiben

Es lasst sich bei der Fulle von Moglichkeiten, die von Bedeutung sein konnten wie etwa Varianten der sehr variablen Innervationsverhaltnisse unter Berucksich tigung der Vagoakzessoriusproblems mechanisch sich auswirkende anatomische Varianten des Kehlkopfbaues, Muskelkontrakturen u a m vorlaufig nur dieses oder jenes vermuten, aber nicht beweisen, oder auch nur ausreichend wahrscheinlich machen

LITERATURVERZEICHNIS

ARNOLD G E Zur Frage der Stimmlippenstellung bei Recurrenslahmung Mschr Ohrenheilk *92* 235 (1958)

FRENZEL H. Pragmatische Systematik in der Hals Nasen-Ohrenheilkunde Munch Med Wschr 1956 972

HOFER G und J JESCHEK s bei JESCHEK J Theorie und Klinik der Stimmbandlahmung Arch Ohr Nas Kehlk Heilk. *162* 237 (1952)

KREJCI F Zur Genese der Stimmbandlahmung nach Jugularisligatur Mschr Ohrenheilk *85* 181 und 316 (1951)

HOFER G Stellungnahme zu Krejci Ebenda 315

KECHT B Krit sche Betrachtungen uber den Ablauf der Kehlkopflahmungen. Mschr Ohrenheilk. *92* 44 (1958)

operation by LEROUX ROBERT, which shows bilateral laryngocele with laryn geal carcinoma, no laryngectomy was carried out in this case

PIETRANTONI has probably had the most extensive experience, but his cases — remarkably — were discovered, not before or during the operation but subsequently, in the course of an aimed study of tomograms of the larynx obtained in a few hundreds of cases, in a small number of cases the laryngeal specimen was further examined CASADESUS also found a laryngocele in a surgical specimen, as did WAYOFF and PERRIN

While a search is necessary to detect an internal laryngocele, an external laryngocele is as a rule also overlooked at laryngectomy, either due to ignorance of the occurrence of these laryngoceles or due to the extremely lateral localization, as a result of which the pedicle is cut unnoticed. This is the case particularly in narrow field operations

To find a laryngocele, one must be laryngocele minded

PIETRANTONI studied the tomograms of 857 cases of laryngeal carcinoma for signs of laryngocele, he found an aerocele" in 53 cases Thirty-one specimens could be examined in detail, with the following results

a An extensive supraglottic carcinoma as a rule existed

b A bilateral laryngocele was found in 12 of the 31 cases, although the X rays showed only a unilateral laryngocele In this respect PIETRANTONI remarks that a tomogram often fails to show the existence of a ventricular dilatation' The explanation is that the laryngocele sometimes contains mucus instead of air

In agreement with LEROUX ROBERT PIETRANTONI, HUIZINGA and others it can be maintained that the association of laryngeal carcinoma with laryn gocele is not so uncommon

Two groups can be distinguished namely

Group I *There is clinical evidence of a laryngocele,* an associated laryngeal carcinoma is discovered at operation
In case no 6 of LEROUX ROBERT bilateral laryngocele was diagnosed and a laryngeal carcinoma suspected

Group II *There is clinical evidence of laryngeal carcinoma,* but a laryngocele is found at operation or subsequently in the surgical specimen This group is the largest

An internal laryngocele is of clinical significance in so far as it changes the features of a laryngeal carcinoma and in so far as the symptoms can be entirely ascribed to the tumour After X ray treatment of the larynx, a laryngocele can suggest a relapse

The changes described may afford an explanation of the pathogenesis of laryngoceles On the basis of a study of carcinomata arising in the sinus of Morgagni, MEDA found a few cases of incipient laryngocele, both on the tomograms and at

The following case report from our clinic can be used as a starting point for further comments upon some questions which may arise in the case of laryngocele associated with laryngeal carcinoma

The patient, born in 1909, visited the E N T specialist on 27th June 1961, with a two-month history of hoarseness In the larynx, at the site of the true and false cords on the left, an irregular swelling was found, an ulcer existed at the base of the epiglottis The left half of the larynx showed virtually complete fixation The tomograms showed a swelling of the soft tissues in the region of the left false cord and disappearance of the left sinus of Morgagni The sub glottic space was intact The right sinus was present, with a rarefaction lateral to it laryngocele? A biopsy revealed the features of squamous cell carcinoma At laryngectomy a bilateral laryngocele was found which extended along the greater cornu of the hyoid bone over a considerable distance in lateral direction Both laryngoceles were carefully dissected out, and resected with the larynx The photograph of the laryngeal specimen clearly shows that two very large laryngoceles were present They were filled, not with air but with mucus The internal wall of the laryngoceles contained mucosa (Fig 1) Nine months later the patient developed extensive cervical gland metastases, in spite of a radical neck dissection, he died as a result of metastases of the cervical vertebrae

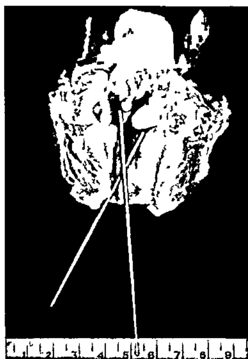


Fig 1 Cancer of the larynx with bilateral laryngocele

The fine specimen shown in Fig 1 with bilateral external laryngocele, proved to be rare although the literature includes illustrations showing laryngeal specimens with unilateral laryngocele (MEDA) and a photograph obtained during an

operation by LEROUX ROBERT, which shows bilateral laryngocele with laryn geal carcinoma, no laryngectomy was carried out in this case

PIETRANTONI has probably had the most extensive experience, but his cases — remarkably — were discovered, not before or during the operation but subsequently, in the course of an aimed study of tomograms of the larynx obtained in a few hundreds of cases, in a small number of cases the laryngeal specimen was further examined CASADESUS also found a laryngocele in a surgical specimen, as did WAYOFF and PERRIN

While a search is necessary to detect an internal laryngocele, an external laryngocele is as a rule also overlooked at laryngectomy, either due to ignorance of the occurrence of these laryngoceles or due to the extremely lateral localization, as a result of which the pedicle is cut unnoticed This is the case particularly in narrow field operations

To find a laryngocele, one must be laryngocele-minded

PIETRANTONI studied the tomograms of 857 cases of laryngeal carcinoma for signs of laryngocele, he found an aerocele in 53 cases Thirty-one specimens could be examined in detail, with the following results

a An extensive supraglottic carcinoma as a rule existed

b A bilateral laryngocele was found in 12 of the 31 cases, although the X rays showed only a unilateral laryngocele In this respect PIETRANTONI remarks that a tomogram often fails to show the existence of a ventricular dilatation" The explanation is that the laryngocele sometimes contains mucus instead of air

In agreement with LEROUX ROBERT, PIETRANTONI, HUIZINGA and others it can be maintained that the association of laryngeal carcinoma with laryn gocele is not so uncommon

Two groups can be distinguished, namely

Group I *There is clinical evidence of a laryngocele,* an associated laryngeal carcinoma is discovered at operation
In case no 6 of LEROUX ROBERT bilateral laryngocele was diagnosed and a laryngeal carcinoma suspected

Group II *There is clinical evidence of laryngeal carcinoma,* but a laryngocele is found at operation or subsequently in the surgical specimen This group is the largest

An internal laryngocele is of clinical significance in so far as it changes the features of a laryngeal carcinoma and in so far as the symptoms can be entirely ascribed to the tumour After X ray treatment of the larynx, a laryngocele can suggest a relapse

The changes described may afford an explanation of the pathogenesis of laryngoceles On the basis of a study of carcinomata arising in the sinus of Morgagni, MEDA found a few cases of incipient laryngocele, both on the tomograms and at

examination of the laryngeal specimen These cases could support MARSCHIK's original view that a laryngocele is a symptom of retention (valve mechanism?)

This hypothesis, however, does not apply to cases of unilateral laryngeal carcinoma with bilateral laryngocele

These cases point in the direction of the views of *LINDSAY*, *LEBORGNE* and others, who mention increased intratracheal pressure as the cause Because some patients with disturbed vocal cord function resort to the use of the false cords — dysphonia plicae ventriculares (JACKSON) — increased intratracheal pressure can in fact cause increased pressure in the sinus of Morgagni when the false cords are occluded

Indirectly, therefore, the carcinoma is the cause of the formation of laryngoceles

The laryngoceles are too frequent to warrant great enthusiasm for a congenital theory

HUIZINGA rightly pointed out, therefore, that a knowledge of the above described combination of laryngeal carcinoma and laryngocele is of great importance to the clinician

REFERENCES

1 BROYLES E. N (1959) Annals of Otology etc *68* 461
2 CASADESUS, J (1951) cit. J Leroux Robert (1957)
3 HUIZINGA, EELCO (1959) Ned T v Geneesk *103* I, 12
4 LEBORGNE, F (1940) Arch Otolaryng *31*, 419
5 LEROUX ROBERT, J (1957) Annals d O.R L *74*, 885
6 LINDSAY, J R (1940) Annals of Otology etc *49*, 661
7 MARSCHIK, H. (1927) Zentralbl. H.N O *10*, 104
8 MEDA P (1952) Arch. Otolaryng *56*, *512*
9 PIETRANTONI, L (1959) Annals of Otology etc *68*, 100
10 SCHALL, LEROY (1944) Annals of Otology etc. *53* 168
11 WAYOFF M and C PERRIN (1961) Journ franç O R L *10* 599

GEDANKEN ÜBER DIE CHRONISCHEN ENTZÜNDUNGEN DER RESPIRATIONSSCHLEIMHAUT

von

B GUSIĆ
Zagreb, Jugoslawien

Wenn ich ein Paar Worte zu Ehren meines Freundes Professor E Huizinga widmen soll, so möchte ich gerne einige Gedanken an ein gemeinsames Gespräch anknüpfen, das wir vor einigen Jahren an den schönen Plitwizer Seen in Kroatien geführt haben und zwar über die chronischen Entzündungen der Respirationsschleimhaut im Allgemeinen und ihre Reaktionsmöglichkeiten im Besonderen. Heute, wo die Untersuchungen gerade an der Respirationsschleimhaut im vollen Gange sind, dürfen vielleicht diese Gedanken auch für einen breiteren Kreis der Kollegen von Interesse sein und darum ihre Erwähnung hier seine Berechtigung finden

Wir sind noch aus der morphologischen Betrachtungszeit aus gewohnt bei chronischen Entzündungen der Schleimhaut des Respirationstraktes eine hypertrophische und eine atrophische Form zu unterscheiden, ja man spricht sogar nach Kretschmerschers Auffassung, von zwei besonderen Arten der entzündlichen Erkrankung einer produktiven und einer solchen mit exudativ alterativen Reaktion Diese Auffassung had Wittmaack weiter ausgebaut Von dem morphologischen Bilde dieser Schleimhaut bei verschiedenen Individuen ausgehend, hat dieser Forscher dann seine bekannten 4 Formen der Mittelohrschleimhaut aufgestellt, die dann mutatis mutandis auch auf der Respirationsschleimhaut der oberen Luftwege ihre Anwendung gefunden haben Den Gewebsaufbau mit der Konstitution gleichstellend hat er die endgültige Schleimhautform von den Umwelteinflüssen abhängig gemacht Andererseits wieder hat W Albrecht und seine Schule auf die genotypische Anlage der Schleimhaut als das Grundelement ihrer weiteren Formenbildung hingewiesen Diese umfasst die Gesamtheit der Anlagen die ein gewisses Individuum von vornherein besitzt Von der Entwicklungspotenz der einzelnen morphologischen Teile hängt in erster Linie die Reaktionsweise der ganzen Schleimhaut auf die auf sie einwirkenden Umwelteinflüsse ab Nicht die Peristase sondern die anlagebedingte funktionelle Variabilität die Entwicklungspotenz einzelner Bestandteile der Schleimhaut, bildet die formbildende Kraft Folglich ist auch der Pneumatisationsvorgang genotypisch vorausbestimmt, wobei eine aktive Pneumatisationspotenz der Schleimhaut vorausgesetzt wird

Ich glaube dass der komplizierte Vorgang der Reaktionsweise der Respirationsschleimhaut auf bestimmte chronische Reize noch komplexer angesehen werden muss Durch klinische, histologische und bakteriologische Untersuchungen die ich im Laufe der Jahre an Tausenden von Patienten aller Altersgrade und beiderlei Geschlechtes durchführen konnte, in einem Lande, wo in den früheren Jahren Ozaene Sklerom, endemischer Lues und Lepra sehr verbreitet waren und es teil

weise auch heute noch sind und wo in der letzten Zeit, nach dem zweiten Weltkrieg, als Folge der schnellen Industrialisierung die industriebedingten Noxen noch dazugekommen sind, haben sich gewisse Anschauungen und Begriffe gebildet die diesen Entwicklungsvorgang vielleicht etwas modifiziert, aber wie ich glaube naher der Wahrheit erscheinen lassen

Wenn wir von anlagebedingten Faktoren ausgehen wollen, so mussen wir, was die Respirationsschleimhaut anbelangt, 4 grundlegende Elemente unterscheiden das Epithel mit seinen Drusen, den bindegewebigen Grundstock, das Gefaßsystem und die Innervation Es ist, glaube ich, ohne weiteres klar, dass das aktive Mesenchym als der entwicklungsgeschichtlich alteste und sehr wahrscheinlich auch am wenigsten hochdifferenzierte Teil in seiner Reaktionsweise am meisten dem des Mesenchyms in anderen Teilen unseres Korpers entspricht oder wenigstens nicht stark von dieser abweicht Anders steht es mit dem Epithel, das in seinen Flimmerzellen eine fur jetzt hochste Differenzierung erlangt hat Und doch, obzwar seine einzelnen Zellen gegen die Einwirkung ausserer Noxen sehr empfindlich sind, und in diesem Kampf bald untergehen, ist ihre Regenerationsfahigkeit staunenswert, wie dies die neuesten Untersuchungen gezeigt haben Dass dabei das Mesenchym als der widerstandsfahigere Teil in dieses Spiel hineingreift und in starkerem Masse den Kampf des empfindsamen Epithels unterstutzt, ist begreiflich Es ist naturlich klar, dass die Reaktionskraft nicht ausschliesslich von der Peristase abhangt, sondern dass sie auch anlagebedingt ist Nur kann man, glaube ich, dabei nicht von einer eigenen Konstitution oder besonderem Genotypus der Respirationsschleimhaut ausgehen, sondern vielmehr von solcher des Mesenchyms oder des Epithels eines bestimmten Organismus im Allgemeinen Ob man dabei von einer Potenz der Entwicklungsfunktionen nach M Heidenhain sprechen kann, oder von einer biologischen Wertigkeit nach der Ausdrucksweise von W Albrecht, bleibt dahingestellt Es hat schon M Schwarz richtig hervorgehoben dass diese Potenz der Schleimhaut nicht allein auf lokalen Abwehrfaktoren beruhen kann, sondern dass dabei auch noch die verschiedensten Gegenspiele allgemeiner Art eine wichtige Rolle spielen Gerade das haben unsere neuesten Untersuchungen uber die Verhaltnisse der Respirationsschleimhaut zum hepatorenalen System hinlanglich bewiesen Wenn wir die Form eines Organes, und das ist, glaube ich, einzig richtig, als Folge seiner Funktion auffassen, wenn wir also Funktion und morphologische Zusammensetzung gleichwertig nebeneinanderstellen, wie es schon W Albrecht getan hat dann mussen wir dieselbe Gleichstellung nicht nur fur normale physiologische, sondern auch fur pathologische Prozesse annehmen Schon Kretschmer hat folgerichtig geschlossen, dass die menschliche Korperform nichts Starres sondern eine langsam sich formende dauernde Bewegung darstellt, eine ‚festgewordene Funktion' ein greifbarer Niederschlag einer grossen Reihe trophischer Impulse oder lebendig gesteuerter Wachstumsvorgange

Aus Allem hier gesagten geht es klar hervor, dass zwar die Starke der Reaktion eines Organes in unserem Falle der Respirationsschleimhaut von der Intensitat der ausseren Noxe abhangig ist aber das andererseits die Form dieser Reaktion morphologisch bedingt ist Sie hangt folgerichtig von der Potenzmoglichkeit der

einzelnen verschiedenartigen Teile des betroffenen Organes ab, wobei die höher differenzierten Teile früher unterliegen als die entwicklungsgeschichtlich älteren, sicher auch widerstandsfähigeren Aus dieser, wie ich glaube, vollkommen klaren Gedankenfolge erklärt sich die Tatsache, dass wir in der Respirationsschleimhaut keine besondere hypertrophische beziehungsweise atrophische Entzündung haben können, dass es sich bei jedem chronischen Entzündungsverlauf um einen morphologisch und funktionell einheitlichen Prozess handelt, der mit einer Hyperämie anfängt, bei weiterer toxischer Wirkung der Noxe zu einer Exudation und Hyper plasie führt, und zuletzt, wenn auch die Reaktionspotenz des Mesenchyms erschöpft ist, in einer Atrophie endet Es hängt also vom Gegenspiel Noxe Reaktionspotenz ab in welchem Stadium der Prozess zum Stillstand kommen wird

Das Gefäßsystem und die Innervation sind gegen Lebensgewohnheitsänderungen äusserst empfindlich Sie reagieren prompt mit schnell steigender, aber dann noch schneller fallender Funktionspotenz, die dann auch in bestimmten histologisch feststellbaren Veränderungen ihren Ausdruck findet Wir können das in unserem Lande täglich beobachten wo grosse Massen früherer Hirten und Ackerbauer aus ihren Dörfern in die neue Industrie und ihre städtischen Siedlungen gezogen sind Aber auch da hat sich die Reihenfolge der Reaktion auf der Schleimhaut nicht geändert Das Prinzip ist dasselbe geblieben, obzwar die morphologischen Ver änderungen, wenn sie sich einmal ausgebildet haben, vorwiegend das Gefäßsystem und die feine Innervation befallen

Es ist also doch die morphologische Zusammensetzung die den Rahmen für die Reaktionsweise eines Organes auf äussere Noxen bestimmt, es ist aber auch die Peristase, die ständigen trophischen Impulse, die einzelnen morphologischen Ein heiten dauernd treffen, die die Steuerung ihrer Wachstumsvorgänge veranlassen und ihre weitere Formung beeinflussen

LITERATURVERZEICHNIS

1 GUSIC B Neues über die atrophischen Veränderungen der Nasenschleimhaut Monatschrift für ORL 6 346 1958

2 SCHWARZ M Die Schleimhäute des Ohres und der Luftwege Biologie und Klinik. Springer Berlin 1949

STAPES FIXATION BY AN INTRA LABYRINTHINE SEEDLING" NEUROFIBROMA AS A CAUSE OF CONDUCTIVE DEAFNESS IN A CASE OF VON RECKLINGHAUSEN S DISEASE

by

C S HALLPIKE *

London, England

In a short paper published in 1950, Dix and Hallpike referred to an earlier report by Just (1930) of a true conductive type of deafness found to occur in certain cases of acoustic neurofibroma

Evidence which appeared to confirm this finding was derived by Dix and Hallpike from their clinico pathological studies of two such cases seen at the National Hospital, Queen Square

In each of these the presence of a unilateral acoustic neurofibroma was confirmed at operation, while the deafness of the affected ear was apparently of the conductive type as attested by the results of the Rinne test Subsequent histological examination of temporal bones revealed the presence of dense coagula which completely filled the fluid spaces of the labyrinths These were thought to have increased the mechanical impedance of the intra labyrinthine portion of the sound conducting pathway, and so to have brought about the conductive type of deafness

For the Rinne test, which determined the diagnosis of a conductive type of deafness, a hissing noise delivered through a speaking tube was used for the masking of the unaffected ear This was almost certainly inadequate for the exclusion of the artefact of the false negative Rinne and, in a later publication, Dix and Hallpike (1958) emphasised this, stating that since their adoption of narrow band masking for all bone conduction tests, they had encountered no evidence of conductive deafness in a series, exceeding 100, of cases of acoustic neurofibroma

The purpose of the present short communication is to present certain of the findings in a case of deafness due to bilateral acoustic neurofibromata in which the clinical evidence of a true conductive type of deafness could be more clearly substantiated, and for which histological examination of the temporal bones revealed a clear anatomical basis This took the form of a small neurofibroma lying within the vestibule of the left labyrinth and impinging upon the deep surface of the stapes footplate

The patient, a Cypriot male, 23 years of age, was seen on 6/3/50 His chief symptoms were bilateral deafness and tinnitus which had been present for 10 months in the case of the left, and for 5 months in the case of the right ear Some unsteadiness of walking had also been evident

No significant abnormality could be seen on examination of the ears, nose and throat Cochlear function the conversational voice could be heard but not

* *From the Aural Department and the Otological Research Unit of the Medical Research Council, The National Hospital, Queen Square, London, W C 1*

understood at either meatus, and rather better on the left side than the right The Rinne test, performed with a tuning fork of frequency 512c/s, was markedly negative on both mastoid processes Pure tone audiometry showed severe bilateral deafness, and confirmed its conductive character Furthermore, with speech audiometry, speech sounds were noticeably better heard, and their nature appreciated

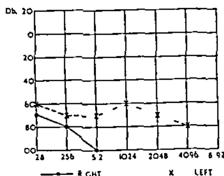


Fig 1 Air conduction audiogram

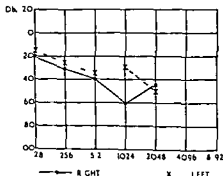


Fig 2 Bone conduction audiogram

by bone than by air conduction Air and bone audiograms are shown in Figs 1 and 2 The bone conduction tests were performed without masking Vestibular function various abnormalities were present including spontaneous and positional nystagmus, derangement of optokinetic and bilateral reduction of caloric nystagmus Clinical diagnosis space occupying lesions in the cerebello-pontine angles bilateral acoustic neurofibromata?



Fig 3 Left temporal bone Low power photomicrograph of horizontal section showing tumor mass in internal auditory meatus Outer ear middle ear and stapes footplate normal Perilymphatic and endolymphatic spaces of the vestibule normal

Surgical exploration of the right cerebello-pontine angle was performed 16 days after our examination and revealed the presence of an acoustic neurofibroma Respiratory failure ensued, and at post mortem examination bilateral acoustic neurofibromata were found to be present with marked distortion of the lower pons and medulla Nodular fibromata were also present on a number of the main nerve trunks throughout the body The temporal bones were prepared for histological examination and serial sections were cut in the horizontal plane Low power photo micrographs are shown in Figs 3 and 4

On the left side (Fig 3) a tumor is shown enlarging the internal auditory meatus The external and middle ears are normal Within the cochlea the neural elements in the modiolus are degenerate with much albuminoid coagulum in the endolymph and perilymph scalae Deep to the stapes footplate the perilymph space and saccule are normal



Fig 4 Right temporal bone Low power photomicrograph of horizontal section showing tumor mass in internal auditory meatus Outer ear middle ear normal. The perilymphatic space of the vestibule contains a small neurofibroma impinging upon the deep aspect of the stapes footplate

On the right side (Fig 4) a tumor mass is seen within the internal auditory meatus The external and middle ears are normal Within the cochlea there is some distention of the scala media with albuminoid coagulum in the scala vestibuli Within the vestibule a remarkable abnormality is present

This consists of a seedling" neurofibroma occupying the greater part of the perilymph space compressing the saccule and impinging upon the deep aspect of the stapes footplate

Discussion

The patholo ical findings make it clear that the case was one of generalised

neurofibromatosis or von Recklinghausen s disease In this condition as described by O Mayer, small seedling" neurofibromata may occur within the labyrinth So far as we know the present finding is unique in that the seedling ', by virtue of its position, must have immobilised the stapes and so provided the anatomical basis of the conductive deafness which was found on clinical examination

The audiological data will now be further considered As shown in Figs 1 and 2, the sensitivity to pure tones of both ears was considerably better by bone than by air conduction This is in agreement with the finding with tuning forks of negative Rinne reactions on both mastoids Although no masking was used for these tests, various circumstances would seem to exclude the possibility of artefact

Thus, for the well known artefact of the false negative Rinne reaction, it is required that the hearing of the opposite ear should be normal or substantially better than that of the ear under test, and should furthermore exhibit a positive Rinne reaction

Upon this basis it could be argued that the negative Rinne reaction obtained upon the right mastoid was a false reaction, with the bone conduction response derived from cross stimulation of the better hearing left ear This would seem to be contra indicated by the fact that the Rinne reaction was also negative upon the left mastoid For this, one explanation could be that in the left ear, too, the deaf ness was of the conductive type But histological examination revealed *no* evidence of any conductive lesion, and some other solution must be sought For this, we turn to the remarkable histological finding of a seedling' neurofibroma within the vestibule of the right labyrinth and the consequent immobilisation of the overlying stapes footplate

In the two bone conduction audiograms shown in Fig 2 the readings for all but one of the test frequencies are substantially coincident It is, therefore, argued that both were derived from the right ear and that the deafness of this ear was in part of the true conductive type with a large bone-air gap

The deafness of the left ear however, was of the perceptive type, and the negative Rinne obtained on the left mastoid was a false reaction with its bone conduction component derived from the right ear

SUMMARY

A clinico-pathological study is presented of a subject with von Recklinghausen s disease and bilateral acoustic neurofibromata Severe bilateral deafness was present The deafness of one ear was in part of the conductive type and attributable to stapes fixation brought about by a seedling neurofibroma arising within the labyrinth and impinging upon the deep aspect of the stapes footplate

ACKNOWLEDGMENTS

For permission to use the neurological and morbid anatomical data included in this report thanks are due to Dr J Purdon Martin Physician in charge of the case and to Professor William Blackwood

REFERENCES

DIX M R and HALLPIKE C. S 1950 Proc Roy Soc Med. 43 291
DIX M R and HALLPIKE, C S 1958 Proc Roy Soc. Med 51 889
JUST T H 1930 Proc Roy Soc. Med 23 722
MAYER O 1917 Zeitschr f Ohrenheilk u f Krankh d Luftwege 75 95

BLOOD VESSELS OF THE OSSICULAR CHAIN

by

C A HAMBERGER, G MARCUSON and J WERSALL *

Stockholm, Sweden

Attempts to study the vascular supply of the ossicular chain by means of the injection technique or direct inspection have failed to reveal the fine architecture of the blood vessels in the mucosa of the middle ear Nager and Nager (1953) followed the vessels in serial sections of human temporal bones, and described the main blood supply to the middle ear Their technique did not, however, allow identification of the fine vessels of the ear Although observations in transparent, injected specimens of human auditory ossicles by Kirikae (1960) demonstrated



Figure 1 Circulation in the human tympanic membrane demonstrated with Sjostrand's technique for staining of the blood cells Observe the rich network of communicating vessels of small calibre

the extensive network of vessels within them, no account was given of the architecture of the vessels on the surface of the ossicular chain Hamberger and Lindgren (1941) demonstrated an extremely rich network of communicating vessels in the tympanic membrane (Fig 1), utilizing Sjostrand's (1934) method for staining the blood cells This method has the advantage of staining the vessels of both the mucous membrane and the bone without distortion It was therefore

* *From the Department of Otolaryngology Karolinska Sjukhuset, Stockholm*

applied in the present study for depiction of the vascular supply to the ossicular chain

Material and Methods

Temporal bones from adults were fixed in formalin The specimens were rinsed in 60 % alcohol The tegmen tympani was carefully opened, and staining fluid according to Sjostrand was dropped into the tympanic cavity This produced immediate staining of the vessels in the mucous membrane as well as rapid penetration into the bone, staining the vessels within it The tympanic membrane was dissected out from the annulus fibrosus together with the adjacent area of the mucous membrane The ossicular chain was loosened from the walls of the middle ear and the stapes was carefully lifted out, together with the surrounding mucous membrane The whole ossicular chain was then further dispersed in the staining solution, to obtain complete penetration of the dye

Results

The mucous membrane of the tympanic cavity forms several folds around the ossicular chain through which the blood vessels reach the ossicles With respect to the main supply of the malleus and the incus, our findings have verified those of Nager and Nager

The *malleus* is supplied by two main nutrient branches from the anterior tympanic artery One branch, given off from the superior branch of the artery, follows the mucosal fold along the superior malleolar ligament, from the tegmen tympani to the head of the malleus It penetrates the latter through the nutrient foramen, forming a large number of intra-osseous vessels The main malleolar branch leaves the anterior tympanic artery, close to its entry into the tympanic cavity from the petrotympanic fissure It runs along the anterior process of the malleus to the lateral aspect of the neck of the malleus, where it penetrates into the bone through one or two nutrient foramens One branch follows the handle of the malleus as a thin, tortuous nutrient vessel The mucosa of the malleus is supplied by a rich network of communicating branches, coming both from the superior and the main malleolar artery Several of these branches run for a varying distance in the bone of the malleus, and communicate with the nutrient vessels

The *incus* is usually supplied by three arteries One small artery from the superior branch on the anterior tympanic artery enters the superior part of the body of the incus close to its articulation with the malleus One branch passes in a mucosal fold from the posterior branch of the tympanic artery to the incus, where it enters the bone at the base of the short process Another branch — denoted by Nager and Nager as the incudal artery — follows a mucosal fold from the anterior tympanic artery, on the lateral aspect of the incus and ends as the main incudal artery supplying the body and the long process of the incus It enters the bone through a nutrient foramen usually localized close to the base of the long process

Although both the incus and the malleus contain a considerable number of small

nutrient branches, passing in all directions through the bone it is largely vascularized by the dense network of communicating vessels in the mucosa shown in Fig 2



Figure 2 The rich network of mucosal vessels shown in the picture of the incus is specially important for the vascularization of the processes of the incus

The *stapes* differs from the other two ossicles with respect to the type of vessels of importance for its circulation Only the head is supplied by nutrient arteries penetrating the bone, the rest of the stapes being supplied entirely through mucosal vessels The main branches to the stapes are given off from the stylomastoid artery and its connexion with the superficial petrous artery One or usually several, small arteries with a varying course follow the stapedius tendon and run in small mucosal streaks or folds from the facial canal to the posterior limb and the posterior part of the footplate Some of these vessels pass across the mucosal fold between the limbs supplying the anterior limb with ascending and descending branches One fairly large branch runs along the anterior limb to the head of the stapes where it penetrates into a nutrient foramen situated on the lower side of the head and dividing into a fine network of vessels within the bone The footplate, which is normally devoid of vessels is covered by a thin mucosal membrane, containing an extremely delicate network of fine communicating vessels This vascular network also receives a few small branches from

he superior and inferior tympanic arteries reaching the anterior part of the ootplate (Fig 3)



Figure 3 The footplate of the stapes and the crura get their vascular supply from the mucosal vessels seen in the pictures A few vessels pass from one side to the other between the crura

Comments

The ossicular chain is partly supplied by nutrient arteries penetrating the bone, and branching into an extensive network of mucosal vessels Although the arteries are usually well visible in specimens treated by the injection technique, the mucosal network is more easily and more reliably identified by the staining method used in the present study This network is particularly important for vascularization of the processes of the incus and malleus, as far as the stapes is concerned it is the only source of blood to its footplate

In operations on the inner ear all the vessels to the ossicles are susceptible to damage in view of their course through the mucosal folds or along the mucosa, lining the walls of the middle ear This applies particularly to the mucosal network supplying the stapes Mobilization of the stapes — as well as other operations in which the stapes tendon is cut and the footplate mobilized — produces considerable disturbances in the circulation of the stapes and its surroundings Bleeding in the stapedial region is generally attributable to destruction of the arterial supply to the posterior part of the ossicle This circulatory disturbance explains why its

use of the stapes itself is always to be preferred For inserting a foreign body is an unphysiological approach involving a certain risk about which an opinion can only be formed after years of experience

To be able to judge the obtained improvements in hearing acuity, we compared (not until at least three months after the operation) the pre and post-operative threshold audiograms of the first group of 100 patients operated upon unilaterally Only in one case the hearing acuity appeared to have become worse In another case the hearing acuity hardly improved, in all remaining 98 cases, however, there was a substantial gain The following quantities were calculated for each person

1 the average gain for air conduction at the frequencies 250 and 500 Hz,
2 the improvement of the speech hearing capacity, i e the average gain for air conduction at the frequencies 500, 1000 and 2000 Hz,
3 *the change of slope of the threshold curve in the 500—2000 Hz band*

Table I gives a survey of the results achieved for these 98 patients according to the measurements in our audiological department

TABLE 1

	decrease of air bone gap in db	increase of speech hearing cap in db	change of slope in db/octave
maximum gain	55	50	+12 5
medium gain	37,5	31,3	— 5
minimum gain	17,5	8,3	—25

From this table the following conclusions can be drawn In the least favourable case of these 98 patients the air bone gap in the bass part of the tone scale was filled up with an amount of 17 5 db, the speech hearing capacity improving by 8 3 db in the most favourable case these values amounted to respectively 55 db and 50 db

The median values (fifty fifty level) which according to the table, amounted to respectively 37 5 db and 31 3 db, also proved results to be favourable In general the subjective experiences of the patients affirm these findings, with the exception of a small group For the hearing acuity of some patients did improve indeed considerably as far as ambient sounds were *concerned but not with respect* to the spoken word This discrepancy could be explained from the curve of the post operative threshold audiogram which showed in these cases a too steep slope (more than 10 db/octave) in the 500—2000 Hz band As a matter of fact low speech components will produce a masking effect on the higher components in all cases with a slope of 10 db per octave or more causing a decrease in intelligibility [4, 5] Column III of the table shows the change which is brought about in the slope of the thr hold audi gram *the most considerable change in a positive sense being*

12 5 db/octave and in a negative sense 25 db/octave The median value was —5 db/octave In twelve patients the slope appeared to have increased after the operation by 10 db/octave or more as a result of a relatively insufficient improvement of the frequency of 2000 Hz

Consequently we may conclude that when the bass part greatly improves, whereas relatively the hearing acuity in the 1000—2000 Hz octave falls behind, this amelioration is unfavourable for improvement of the speech hearing function As long as it has not become fully clear what this discrepancy in some of these patients is due to, this difficulty cannot be removed

Since the median value in column three indicates that in at least half the cases the slope changes in a negative sense, it is advisable, regarding the prognosis as to the post-operative speech hearing capacity, to take into account the pre-operative value of this quantity in the 500—2000 Hz frequency band Therefore it is our standard procedure to test every patient by means of so-called triplet speech audiometry

Taking account of the necessity of a prolonged audiometric follow up as regards *the post-operative hearing acuity and also considering the possibility of a further* improvement in the operation technique in the near future, up to now we have — after a successful first operation — not attempted to operate on the other side as well

REFERENCES

[1]) PORTMANN, M and CLAVERIE, G Rev Laryng *79* 306, 1958

[2]) *SHEA J J Ann Otol, Rhinol and Laryng 67 932 1958*

[3]) RUEDI, L Pract. Oto-Rhino-Laryng 22 410, 1960

[4]) TASELAAR, M Groningen University thesis, 1959

[5]) VAN GILS J J P Groningen University thesis, 1962

DER RHINOGENE KOPFSCHMERZ UND SEINE DIAGNOSE

von

G. HOFER

Graz, Österreich

Das Kapitel nasaler Kopfschmerz hat eine bis in die letzten Jahrzehnte des vergangenen Jahrhunderts reichende Vorgeschichte. Zu einer Zeit, da die Ursachen für den nasalen Kopfschmerz noch gar keiner eingehenden Beobachtung unterzogen worden waren, machte die Lehre von den nasalen Reflexneurosen grosses wissenschaftliches Aufsehen. *Voltolini* teilte mit, dass es ihm gelungen sei, nach Entfernung einer grossen Anzahl von Polypen, einen Fall von Asthma bronchiale zur Abheilung zu bringen. Der Berliner Kliniker *Bernhard Fraenkel*, der sich vergebens mit der Frage kritisch beschäftigte, kam zu dem Schluss, dass die Verlegung der Nasenatmung allein nicht für das Auftreten des Asthmas verantwortlich gemacht werden könne, es vielmehr nervöse Einflüsse seien, die ihren Ausgang von dem übersensiblen Organ nehmen. Diese Stellungnahme des bekannten Berliner Klinikers 1882, veranlasste *Wilhelm Hack* ein förmliches Neugebäude von pathologischen Zuständen zu errichten, die von der Nase ihren Ausgang nehmen sollten, und zwar von ganz umschriebenen, pathologisch wenig in Erscheinung tretenden Stellen der Nasenschleimhaut. Eine unübersehbare Literatur war bald die Folge der Hack'schen Untersuchungen, von denen besonders die Mitteilungen von *Wilhelm Fliess* eine Vielzahl nasaler Reflexneurosen und Nasenfernerkrankungen glaubhaft machen sollten.

Störungen der weiblichen Genitale mit allen ihren Folgen, Neuritiden des Trigeminus, der Nerven von oberen und unteren Extremitäten, Zirkulationsstörungen, *Kopfschmerz und Migräne*, Harn- und Nierenerkrankungen, Störungen im Sehvermögen, Augen-, Ohren-, Geruch- und Geschmackstörungen, ja sogar psychische Störungen, endlich Hauterkrankungen sollten allesamt Folgen nasaler Affektionen sein und als Reflexerkrankungen gewertet werden dürfen.

Noch im Jahre 1930 kam eine Schrift des Berliner Professors *Koblank* heraus, der sich restlos der Hack-Fliess'schen Theorie anschloss, wobei er als Ursache für die in der Nase auftretenden Anomalien gesteigerte Sexualzustände verantwortlich machte, als deren Folge dann nasale, reflektorisch ausgelöste Erkrankungen auftraten.

Das interessante Kapitel der nasalen Reflexneurosen, es konnte ja nicht ausbleiben, fand scharfe Kritik und in späterer Zeit unbedingte Ablehnung durch eine Reihe namhafter Rhinologen und Neurorhinologen (*Kuttner, Hajek, Waget* u. v. a.).

Der Tenor dieser Ablehnungen konzentrierte sich als eine einfache Formel: Es gebe keinen Fall einer nasalen Reflexneurose, die nach endonasaler Operation *dauernd* geheilt bleibe, wenngleich Stillstände bei allen schweren vegetativen Neurosen durchaus öfter vorkommen.

Der Eingriff an der hypersensiblen Nasenschleimhaut nimmt keinerlei gesonderte Stellung ein gegenuber einem solchen an anderen sensiblen Korperstellen Das Facit ist der Eingriff an einer hochsensiblen Korperstelle an sich und ist nicht beschrankt fur die Nasenschleimhaut in Geltung

Mittlerweile war die Forschung uber Ursache und Verlauf der Erkrankungen der Nebenhohlen der Nase so weit gediehen, dass der rhinogene Kopfschmerz als Kardinalsymptom der Nebenhohlenerkrankungen erkannt und diesem die ent sprechende Wurdigung eingeraumt werden konnte

Welcher Art sind nun diese *rhinogenen Ursachen fur den Kopfschmerz*

An erster Sellte stehen die entzündlichen Erkrankungen und Eiterungen der Nasennebenhohlen Dabei ist die Entzundung der Schleimhaut selbst und die all fallige Retention von Sekret in der entzundlich erkrankten Nebenhohle, die die Ursache fur den Kopfschmerz abgeben Es bedarf also zur Sicherung der Diagnose unter Umstanden lediglich des Nachweises der Entzundung an sich Dass der Kopfschmerz, wie wir wissen, meistens als Tiefenschmerz empfunden wird, liegt dabei wohl in dem Umstand, dass, wie die Erfahrung und das Experiment lehren, der Trigeminusschmerz meistens in jenem Teil des Nerven empfunden wird, der als der empfindlichste Teil des Nerven erkannt ist (Ramus meningeus trigemini)

Neben diesen uns heute am langsten bekannten Ursachen fur den nasalen Kopf schmerz mussten wir in den Untersuchungen besonders amerikanischer Autoren, noch andere Ursachen fur den rhinogenen Kopfschmerz zur Kenntnis nehmen Zunachst den sogenannten Unterdruckkopfschmerz Dieser findet seine Erklärung darin, dass durch Abschluss der Nebenhohlen gegen die Nase, die Luft der ab-geschlossenen Hohle von der Schleimhaut teilweise absorbiert, einen Unterdruck innerhalb der Hohle zur Folge hat, der auf dem Wege des Gefaßsystems zum Kopf schmerz fuhrt Diesen Abschluss besorgt ein hochgradig verbogenes Septum, das durch Kompression der mittleren Muscheln den mittleren Nasengang verschliesst und damit die in demselben gelegenen normalen Öffnungen von Siebbein, Stirn und Kieferhohlen verengt, resp schwer passierbar macht

Atiologisch kommt weiter ein Moment fur den rhinogenen Kopfschmerz in Frage, namlich das Bestehen von breiteren oder engst umschriebenen Kontakt punkten und Druckpunkten einander gegenuberliegender Teile in der Nase

Mit dieser Erfahrung ist die alte Hack Fliess'sche Lehre von der *Fernwirkung* nasaler pathologischer Zustande in sehr partieller Form zu einem beschränkten Recht gekommen Die Tatsache dass solche Kontaktpunkte in der Nase etwa zwischen Septum der unteren Muschel usw beseitigt, den Kopfschmerz dauernd verschwinden lassen ist ein Argument fur die Annahme des Bestehens eines so benannten Kontaktkopfschmerzes fur dessen Zustandekommen teilweise noch ver schiedene Erklarungen (Trigeminus) gegeben werden

Ausgehend von den oberen Luftwegen und *nur im ubertragenen Sinne, finden* sich Kopfschmerzen bei adenoiden Vegetationen und chronischen Adenitiden des Tonsillarsystems Die Lokalisation Ersterer ist besonders am Hinterhaupt

Fur die ersteren Typen werden zirkulatorische Ursachen mit ihrer Verbindung mit intracraniellen Alterationen verantwortlich gemacht (siehe die Untersuchungen

von *Citelli* uber Verbindung von extra und intracraniellen Teilen der Hypophyse (Rachendachhypophyse)), daneben werden aber auch infekttoxische Noxen infolge entzundlicher Prozesse am Adenoidengewebe von Gaumen- und Rachendach in Erwagung gezogen

Zur Differentialdiagnose diene die Erkenntnis, dass folgende Erkrankungen ganz allgemein Kopfschmerz auslosen konnen

1 Raumbeengende Prozesse in der Schadelhohle, 2 Entzundungen der Hirnhaute, 3 Systemerkrankungen des Zentralnervensystems, 4 Augenerkrankungen, 5 Ohrenerkrankungen, 6 Syphilis in ihren verschiedenen Formen, 7 die Arteriosklerose der Gehirngefasse, 8 Herz und Lungenerkrankungen, 9 Magen Darmerkrankungen, 10 Nierenerkrankungen, 11 Zahnerkrankungen, 12 Anamie und Chlorose 12 Hyper und Hypotonie, 13 Prodromaler Kopfschmerz, 14 *neurasthenischer* Kopfschmerz (neurovaskular), 15 chronische Intoxikationen, 16 Migrane, 17 rheumatischer Schwielenkopfschmerz, 18 Kopfschmerz bei Frauenerkrankungen

Nach dieser Übersicht ist es klar, was bei einem Patienten, dessen Kopfschmerz als nicht „nasal" erkannt worden ist, zur Differentialdiagnose erhoben werden muss

1 Die genaue Erhebung der Anamnese 2 interne Untersuchung, 3 Augen und Ohrenuntersuchung, 4 Blutuntersuchung und Blutdruck etc, 5 neurologische Untersuchung 6 Harnuntersuchung 7 Sero Reaktion, 8 Gynakologische Untersuchung

Aus einer langjahrigen eigenen Erfahrung schien mir diese differential diagnostische Analyse zur Klarung des Krankheitsbildes notwendig zu sein und dass sich kaum ein Fall der richtigen Diagnose verschliesst wenn diese Analyse exakt durchgefuhrt wird

PRE AND POSTOPERATIVE ENDOSCOPIC PROBLEMS ASSOCIATED WITH CARDIOVASCULAR SURGERY

by

PAUL H HOLINGER *
Chicago Illinois, U S A

FOREWARD

Professor Huizinga, who is honored by this volume prepared for his 70th Anniversary, has contributed richly to the field of peroral endoscopy He has maintained a keen interest in the advance of thoracic surgery that has occurred during his medical career and has been most enthusiastic in supporting and correlating the endoscopic and surgical studies of diseases of the respiratory tract Outstanding examples are his descriptions of bronchographic techniques and demonstrations of bronchial anomalies that provide accurate reference to these phases of bronchology [1] It is a great privilege and honor to participate in this salute to Professor Eelco Huizinga

INTRODUCTION

Many of the pre and postoperative problems of cardiovascular surgery are of special endoscopic interest and importance Some are the result of the cardiovascular abnormalities themselves, others are the results of the surgical procedures designed to correct them This brief review summarizes clinical experience in this field, limitation of space prevents detailed documentation

THE LARYNX

Major congenital anomalies are frequently associated with anomalies in other areas A high incidence of laryngeal anomalies is associated with congenital heart disease and these accentuate respiratory embarrassment and cyanosis Cysts, laryngoceles, webs and laryngeal paralyses are found more frequently than in otherwise normal infants and often the laryngeal anomaly creates sufficient obstruction to tip the cardiopulmonary balance into decompensation These anomalies must be recognized by direct laryngoscopy but delay in diagnosis results from the clinical impression that the cyanosis or a weak breathy cry are due to the heart lesion or the weakness of the cardiac patient rather than to an obstruction produced by a laryngeal anomaly

Unilateral left cord paralysis is often a preoperative finding in vascular rings,

* From the Departments of Otolaryngology and Bronchology The University of Illinois College of Medicine and the Children's Memorial Hospital Chicago Illinois U.S.A

Later in the clinical course of the cardiac patient bronchiectasis of the left lower lobe is encountered in more than a normal incidence This appears to be the end result of the compression of the left bronchus by the enlarged heart If bronchiectasis is developing repeated aspiration antibiotics and chemotherapy will aid in controlling infection but digitalization and other measures must be instituted to reduce cardiac size to relieve bronchial compresson If bronchiectasis is well established lobectomy is essential to improve the patient s total medical status

REFERENCES

1 HUIZINGA E and SMELT, G J Bronchography Van Gorcum and Co Assen Nether American Otological Society

2 POTTS W J HOLINGER P H and ROSENBLUM A H Anomalous Left Pulmonary Artery Causing Obstruction to Right Main Bronchus — Report of a Case J A M A Vol 155 (Aug) 1954 1409 1411

3 MALONEY W H. Peroral Endoscopy in Cardiac Surgery Tex State Jour of Med 49 861—864 (Dec) 1953

4 HOLINGER P H. Clinical Aspects of Congenital Anomalies of the Larynx Trachea Bronchi and Oesophagus The Semon Lecture 1960 The Journal of Laryngology and O ology (Jan) 1961 Vol LXXV 1—44

PAEDO AUDIOLOGY — A FACET OF MODERN SPECIALISM

by

H C HUIZING and P E HOEKSEMA *

Groningen The Netherlands

When in 1940 Eelco Huizinga entered his professorship — the subject of his inaugural speech being Modern specialism' — there were few indications yet that otology had passed the threshold of a most important evolution

Undoubtedly one of its facets is *paedo audiology*, a branch of science that has aroused great interest during the last fifteen years and to which he devoted much attention As a matter of fact the historically grown relationship between two ancient Groningen Institutes our University and the Royal Institute for the Deaf may chiefly account for this

It was the cause that in 1943 attempts were initiated for tracing hearing remnants in deaf children and for determining by means of audiometry the degree of residual hearing

In this year — thanks also to the cooperation of Drs Buchli, director of the Royal Institute for the Deaf — we succeeded for the first time in making reliable threshold audiograms on deaf pupils, which in some cases referred to children only four years old In this way an early selection of young children with and without usable remnants became possible

In the same period, owing to all kinds of technical inventions of war industry, the basis was laid for the development of the modern, wearable hearing aid

It has been proved that it is the constant wear close to the mouth, of the wearable hearing aid which plays such a fundamental part in our acoupedic treatment of the deaf child with residual hearing

The fact that this handicapped child is enabled to learn to hear itself through apparatuses — at first consciously, later also without being aware of it — means a new approach The child then becomes able to build up an auditorily controlled voice and may acquire an articulatory basis from which understandable speech emerges This all has led to a very important progress on the road to prevention of deaf mutism It s true that in this matter we are concerned with a so-called secondary prevention but nevertheless the consequences of deafness are mitigated in a considerable degree

We have to do here with the fundamental principle of as good as possible a recovery of a series of undeveloped cybernetic processes, which play an important part during the voice formation period already

A normally developed voice is characterized among other things by a natural rising and falling intonation whereas the voice of the deaf has a strikingly monotonous character The auditory emotional aspect is lacking That is the reason why the speech of the deaf misses a functionally most important factor For in connected texts the rise and fall in the voice pitch contributes in a con

* *From the Department of Audiology of the Oto-Rhino-Laryngological Clinic of the University Hospital at Groningen*

siderable degree for concept formation on the part of the audience If the sentence melody is lacking it becomes much more difficult to grasp the content of speech

In the babbling period already the normal hearing child learns to control his voice and instinctively he will practise these modulations while his surroundings provide him with the right patterns Hearing is the only sensory organ that enables him to verify whether his attempts at imitation are successful or not

This controlling function of hearing is possible owing to the feed back phenomenon by which a small part of the vocal energy is transmitted to the sense of hearing partly via the outer ear canal partly by way of bone conduction more directly to the cochlea

The auditory organ being highly sensitive, a very small part of the vocal energy is sufficient to enable the ear to exercise a monitoring function

This so-called feed back mechanism causes, by way of the auditory organ a mechanical acoustical neurological loop This makes it possible for the ear to develop into an error-controlling organism This control function mainly requires a learning capability Thus it becomes clear in what way a, from a physiological point of view, inferior function — that of babbling — may pave the way for a physiologically much more superior function the guiding and controlling so-called cybernetic function This is possible thanks to the baby's everyday vocal play and the opportunity to imitate what his daily surroundings offer

Every growing up child which is handicapped by a serious perceptive impairment and therefore has an equal loss for both air and bone conduction lacks in a more or less degree the above mentioned auditory controlling mechanism which is indispensable to make every act of speaking into a fluent melodical and rhythmical psychophysical process

The principle of this new type of auditory training of the central nervous system we practised first in the forties with high fidelity group amplifiers It soon appeared however, that the individually adjustable wearable aid was to be the adequate solution for advancing the auditory cybernetic processes in the congenitally deaf child

When in 1947 for the first time satisfactory results with wearable hearing aids had been obtained in some hard of hearing children in 1950 a number of three year old deaf pupils of the *Royal Institute for the Deaf* at Groningen with usable hearing remnants were fitted out with such an apparatus After a few months already such favourable results were obtained that it was decided that every child with residual hearing however slight would be fitted out with a wearable hearing aid

Since that time much attention was devoted to an early tracing of the deaf child so as to prevent deaf mutism

Since 1955 a new start was made with the introduction of so-called hometraining This means the use of a hearing aid in domestic surroundings by children of ca 12 months and older their mother having been instructed about use and management Many dozens of early traced deaf children have been disclosed acoustically for the last few years through home training

The aims easiest obtainable are in the first place to awaken the child for the everyday ambient sounds and in the second place to give him the opportunity to monitor his own voice auditorily by using the hearing aid. In children with relatively still considerable hearing remnants a spontaneous speech development can be expected after some years.

The photograph printed here shows a group of mothers of deaf children gathered in our clinic for a four days instruction course.



Instruction course for mothers — June 1959

Quite another aspect of paedo-audiology refers to the case of the hard of hearing child. In these cases we are concerned with a much slighter handicap.

Audiological school-examination has indicated that the number of children impeded in their intellectual development by a hearing impairment is much larger than was surmised. A mass investigation of 10 000 school children performed at the initiative of our clinic showed that about 4 % of the school going children is more or less handicapped in class. It appears that in a great number of these cases the child's situation can be improved by medical treatment, in other cases audiological revalidation ought to be considered. As this concerns large numbers, various measures are necessary and in the first place those which aim at tracing the hearing handicapped child.

Both for the deaf and for the hard of hearing child it is of the utmost importance that as early as possible parents and educators make certain about a possible loss in the child's hearing acuity. Only when one is sure about that adequate measures can be taken. The realization of academical audiological centres in our country is an important facet in the evolution of this specific example of modern specialism.

The fact that we could describe this single aspect from the rich bouquet of successful scientific progression in the Groningen clinic during Huizinga's term of office tends to make us very grateful for his inspiring leadership. This has meant very much indeed to his daily surroundings.

It is sometimes said that les hommes ont deux amours leur patrie et Paris, but Eeko Huizinga still had a third one his clinic, in which he carried on for over fourty years from dawn to dusk.

ON THE EXAMINATION OF THE FUNCTION OF THE OTOLITHS

by

L B W JONGKEES *

Amsterdam, The Netherlands

The examination of otolithic function has been a difficult one not only in the clinic but also in the laboratory In the beginning of this century a lot of unnecessary difficulties complicated matters as it was not understood that no such thing as a difference between dynamic and static stimuli could exist It has been clearly proven that there is no fundamental difference in vestibular stimulation by linear accelarations as represented in dynamic static or centrifugal forces (JONGKEES, 1944, JONGKEES and GROEN, 1946), neither qualitatively nor quantitatively

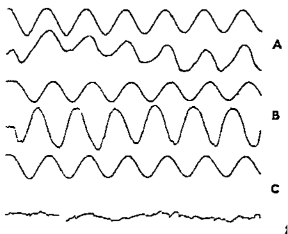


Fig 1 The effect of linear accelerations on the parallel swing upon
A a normal subject
B a patient after unilateral labyrinthectomy
C a patient with two dead labyrinths
Upper curves movements of the swing
Lower curves eye movements

Labyrinthine reflexes, we stated, can be divided in those provoked by rotatory accelerations acting upon the semicircular canals and others caused by linear accelerations acting upon the otoliths In a series of experiments upon humans subjected to both forms of acceleration, this view was shown to be highly probable

The examination remained a delicate and difficult affair as there was no simple objective way to measure the *effect* of the stimulation The sensation of displace

* *From the Department of Oto Rhino Laryngology University of Amsterdam (Director Prof Dr L B W Jongkees)*

ment provoked in the test subject by the cooperation of a horizontal linear acceleration and gravity could be used in scientific experiments (JONGKEES and GROEN, 1950) but it was much too complicated for routine clinical use

On the other hand compensatory eye movements can also be measured but the technique required for this was difficult and time-consuming (JONGKEES, 1953) BENJAMINS and HUIZINGA (1927) proved that the counterrolling of the eyes of pigeons disappeared after destruction of the saccule VERSTEEGH (1927) found that this movement was still partly intact after severing the utricular nerves in rabbits

The technique of stimulation of the linear part of the labyrinth was another

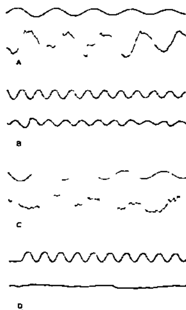


Fig 2 The action of rotatory (torsion swing, A and C) and linear (parallel swing B and D) accelerations
Upper curves the movements of the swings
Lower curves eye movements
A and B before operation C and D after total labyrinthectomy on the left and destruction of the otoliths on the right side

difficulty In lifts, on fast lorries or in complicated positioning apparatus, the examination was possible but too complicated The introduction of the parallel swing (JONGKEES and GROEN, 1946) with electronystagmography has made it possible to study these otolithic eye reflexes more easily and more accurately than before (PHILIPSZOON, 1959, JONGKEES and PHILIPSZOON, 1960) The sinusoidally changing linear accelerations acting upon the animal or patient cause eye movements with a similar sinusoidal rhythm After destruction of one

labyrinth they are still present, but they cannot be elicited any more after destruction of the two labyrinths

In order to prove that it is really the otolithic part of the labyrinth that provokes these reflexes, PHILIPSZOON performed a series of operations upon rabbits He destroyed the entire labyrinth on one side, on the other side the sacculus and the utricular nerve were destroyed after the technique of VERSTEEGH (l c) Histological control (JAMES) showed the operations to be complete in 8 rabbits Before the operation all the rabbits showed the normal pattern of changing eye positions upon the parallel swing, afterwards no more trace of the action resulting *from the swinging* could be found *in the recordings* The *reactions to rotatory* stimulation on the torsion swing (v EGMOND, JONGKEES and GROEN, 1949), however, remained quite normal

I consider this sufficient proof that the parallel swing is a simple instrument to stimulate the otoliths, and only the otoliths Combined with electronystag mography its use is the only easily practicable method to investigate otolithic function in patients by recording the compensatory eye movements due to stimu lation with linear accelerations of sinusoidally changing form

SUMMARY

The parallel swing is a simple device to examine the otolithic function if combined with electronystagmography

REFERENCES

BENJAMINS, C E and HUIZINGA, E. Pflug Arch ges Physiol *236*, 59, 1937

v EGMOND, A A J, JONGKEES, L B W, GROEN, J J J Physiol *110*, 1, 1949

JAMES, J Pract Oto-Rhino-Laryng 1962 (in the press)

JONGKEES L B W N T v Geneesk *88*, 198, 1944
Fortschr Hals-Nasen Ohrenheilk *1*, 21, 1959

JONGKEES L B W and GROEN, J J J Laryng *61* 529, 1946
J Laryng *64* 135, 1950

JONGKEES L B W and PHILIPSZOON A J Acta Phys Pharm Neerl *9*, 240, 1960
Acta Phys Pharm Neerl *10* 239 1962

PHILIPSZOON A J Thesis Amsterdam 1959
Pract Oto-Rhino-Laryng. *24*, 193 1962, idem in the press
N T v Geneesk *106* 479, 1962

VERSTEEGH, C *Acta Oto-Laryng 11, 393 1927*

THE INCUNABULA OF TELEPHONIC AUDIOMETRY

by

GEORGE KELEMEN *

Boston, Mass, U S A.

The invention of the telephone by Alexander Graham Bell (1847—1922) carries the date 1875 The first transmission of sound by Bell took place on June 2, 1875, he switched from batteries to magnets December 1876, and applied for a patent in 1877, this was granted the same year

Cordia C Bunch (1885—1942), the historian of audiometry stated in 1941 " the word audiometry today is restricted to instruments in which the stimulus tones are generated electrically This being the case, the invention of the telephone by Alexander Graham Bell places a definite limit upon the search for the origin of the modern audiometer "

The invention was appropriated very soon to determine hearing power as shown by communications from the years 1878 and 1879 They are called, here, "incunabula" everything that appeared before 1880 Table 1 lists 10 names 5 belonging to physiologists or physicists, and 5 to clinicians Collaboration of this nature never ceased and proved to be most beneficial This tabulation is given to round out previous incomplete records

TABLE 1

	Lecture	Publication
Hartmann	Jan. 11, 1878	1878 (Apr 30)
Hoegyes	Apr 12, 1878	1878
Tarchanoff		1878 (Nov 4)
Wodtke		1878
Preyer	Feb 21 1879	1879
Hoegyes	Apr 4, 1879	1879
Richardson	May 15, 1879	1879
Hughes	May 15, 1879	1879
Koerting		1879 (July)
Hospitalier		1879 (June 15)
Green	Dec. 30, 1879	1880 (Nov 11)

It is a credit to the alertness of the scientific world of the period that by the end of 1879 an entire group of investigators tried to apply the new concept to the testing of hearing power On the other hand, compared with the impact of

* From the Department of Otolaryngology, Harvard Medical School and the Massachusetts Eye and Ear Infirmary Boston Aided by Grant no 361 169, of the Central Bureau of Research, American Otological Society

another epocal invention, Roentgen's rays, applied to medical purposes within months after discovery and expanding in use ever since without interruption, acceptance of the telephone based audiometer suffered a long refractory" period

The apogee of the present was reached with the advent of surgical interventions against deafness , Si les audiogrammes et les audiometres firent la fortune de Lempert, on peut dire qu'il fit la fortune des audiometres" (Hicguet, 1947) This was parallelled by the requirements of the rapid build up of communication, or information, theory Yet even today, a near-century after the discovery of the telephone by Bell and 125 years after the observations of Page at Salem, the ideal audiometer has not been found, according to the testimony of the discussers of Huizing's report at the first international conference of audiology, 1948 Huizinga (1957) may have been on the right beam with his statement that information is a conception of a subtlety far beyond that which can be expressed in mathematical formulae"

THE EAR, NOSE AND THROAT SPECIALIST AND THE TUBERCULOSIS CLINIC

by

J K KRAAN *

Haren, The Netherlands

During the last decennia the task of the ear, nose and throat specialist in the tuberculosis clinic have undergone a thorough change Up to 1945 he was con sulted in cases of patients who suffered from tuberculosis of one of the organs with which his specialism is defined Usually this was not a very thankful task because in the greater part one was concerned with affections that could be cured hardly or not at all

In most cases it was very difficult to treat the tuberculous inflammation of the ear because the necessary operative therapy involved a considerable risk No effective drugs existed for infections with non tuberculous bacteria and for tuber culosis itself Often an awaiting attitude was adopted and most patients kept a chronic purulent ear process Nor did lupus of the nose offer an opportunity for scoring a great success Also when this chronic skin tuberculosis had come in a rather inactive stage, the possibility of a plastic operation with varying success remained for only a very small group

Finally as regards tuberculosis of the bronchial tubes diagnostics mostly had to be confined to larynx and trachea Only seldom did one venture to examine with the first bronchoscopes the main bronchi and the lobe bronchi The chest physician s fear for complications kept the performancy of bronchoscopy too much in check

Moreover the nature of the aberrations in the bronchial tubes was often so serious that there was little chance of a satisfactory therapeutic result afterwards The older laryngologists know too well the wide-spread destructive tuberculosis of larynx and trachea When in the laryngoscopy one found, apart from a diffuse swelling and redness some superficial ulcera this gave cause for gratification already Recovery with more or less extensive scar retraction was possible when lung tuberculosis was cured

Besides we knew the comprehensive processes involving destruction of the vocal cords and the cartilage of the larynx We still remember many patients in our clinic who were not allowed to speak for weeks Recovery only attended with a hoarse voice was a great success in those years

All these patients have become exceptions nowadays, especially thanks to the drugs

Tuberculosis of the ear has become an exception which is still found sometimes after a miliary dissemination Lupus is cured in a short time particularly nice

* *Sanatorium B atrix oord Haren (Gr) (Director Prof Dr J K Kraan)*

STREPTOMYCIN AND NOT DIHYDROSTREPTOMYCIN

by

L MEYLER

Groningen, The Netherlands

The toxic effects of the streptomycins on the ear consist of vestibular disturbances and loss of hearing

Disturbances of equilibrium

Vertigo may suddenly occur, also it may be preceded by tinnitus and headache for a day or they may develop gradually If serious, vertigo is often accompanied by nausea and vomiting and the patient is dizzy even if he lies down quietly. This period of severe vertigo generally lasts for 7 to 10 days after discontinuance of treatment, but usually recovery is not complete and the patients remain dizzy on sudden movement for a considerable period The vertigo does not disappear by virtue of recovery of the vestibular apparatus, but due to the body having learned to maintain balance in different ways

The slight vertigo initially only develops if the patient moves, and especially on sudden movement of the head and when walking in the dark Walking over uneven ground and descending stairs causes trouble This leads to a feeling of uncertainty Also, the patients complain that they are less able to recognize people and objects when they are walking, everything is seen as if in a haze A certain ataxia may also develop since the patients reach too far when they want to seize objects There is also a tendency to topple over if an arm is extended In children the head wobbles as in the newborn

The objective findings on examination of the vestibular apparatus are not wholly parallel to the complaints of the patient This is due to the capability of the body and especially the eyes to take over the vestibular function

In nearly all patients treated with streptomycin for a long time a markedly reduced or absent reaction of the cristae in the semicircular canals is observed on rotation and during the caloric test Usually this does not disappear

If one immediately discontinues streptomycin when the patient begins to feel dizzy the sensitivity of the vestibular apparatus may partly recover, especially in young people However, it is also possible for the symptoms to aggravate despite discontinuance of therapy

Involvement of the vestibular apparatus is always bilateral The chance of damage developing is greater after the age of 45 Regular examination by an otorhinolaryngologist is desirable

Damage to the vestibular apparatus is related to the daily dose and duration of treatment and therefore to the total dosage of the antibiotic A long term course

with low dosage leads to equal damage as a short term course with a larger dosage

The chance of vestibular damage developing is less when dihydrostreptomycin is used than with streptomycin

Deafness

Deafness is the most serious complication of treatment with dihydrostreptomycin It does not always depend on the total dosage and may occur months after the treatment with dihydrostreptomycin has been discontinued Consequently, in many cases deafness develops without this being attributed to dihydrostreptomycin

How serious the problem is, will be evident by some reports

At the ENT department at Turin 138 cases of deafness caused by dihydrostreptomycin were observed 15 Cases of irreversible hearing loss occurred after administration of only small doses[1]

Glorig reported in a series of 61 cases receiving different doses of dihydrostreptomycin, 14 patients suffered complete deafness In 13 patients the deafness developed only 1—5 months after the termination of therapy[2]

32 Cases of permanent hearing loss due to dihydrostreptomycin observed over a period of 4 years are reported in one publication In many instances this had resulted from the use of quite small doses (1—3 g)[3]

Domon reported that a daily dose of 2 g of dihydrostreptomycin caused tinnitus and deafness in 50 % of the patients The other half developed hardness of hearing and tinnitus[4]

26 Cases of loss of hearing caused by drugs have been reported by Sheehy Twenty were associated with dihydrostreptomycin In 8 patients this antibiotic was used in treatment of a cold![5]

According to Ruedi a o dihydrostreptomycin damages the hearing by the toxic effect on sound perceptive hair cells in the organ of Corti[6]

Damage to hearing due to streptomycin is very rare and has only been observed at dosage levels of 2—3 g daily maintained over long periods

A serious warning against treatment of dihydrostreptomycin and streptomycin in cases of renal insufficiency is necessary These drugs should never be given to patients whose renal function has not been investigated and never on any account when there is severe oliguria

Owing to accumulation, toxic manifestations occur in patients with renal disease after a short course In uraemic patients the recommended dosage is 1 g followed by 0 5 g every two to three days

According to some investigations both vestibular and cochlear damage may be appreciably mitigated when pantothenic acid is added

The risk of impairment of hearing is not diminished when streptoduocin (equal parts of streptomycin and dihydrostreptomycin) is used instead of dihydrostreptomycin alone but the disturbances in the vestibular system are milder when this combination is given instead of streptomycin alone

It is reported that streptomycin alone is better for the organ of hearing than

streptoduocin From an audiological examination of 105 patients it appeared that hearing was affected 3 times more often by streptoduocin than by streptomycin alone Vestibular disturbances however, occurred 5 times less frequently when streptoduocin was employed[7]

For a prolonged treatment the combination offers no advantages over the use of streptomycin alone

Dihydrostreptomycin (but also streptomycin) has been reported to be able to pass the placental barrier There are reports of deafness (Kern[8]) in the newborn baby caused by the administration of dihydrostreptomycin to the mother in the first months of pregnancy Also the vestibular organ may be damaged when the mother to be is treated with streptomycin

Conclusion

Streptomycin is valuable for treating tuberculosis while for other infections it is rarely effective

Dihydrostreptomycin should not be manufactured any longer It has no advantages over streptomycin only serious disadvantages

Combined preparations of penicillin and dihydrostreptomycin should not be manufactured If such a preparation is needed penicillin and streptomycin may be combined Personally I have hardly ever needed to resort to such a combination

REFERENCES

1 ROSSI G CARANDO DAMANIO E DE MICHELLS G and MAGGIOROTTI P Acta med Scand *169* (1961) 169

2 GLORIG A Ann Otol Rhinol and Laryng St Louis *60* (1951) 327

3 SHAMBAUGH G E a.o J Amer med Assoc *170* (1959) 1657 WEHRS R E Journal Lancet *78* (1958) 505

4 DOMON Ch M KILBOURNE Ph C and KING E Q Amer Rev Tuberc *60* (1949) 564

5 SHEEHY J L Calif Med *94* (1961) 363

6 RUEDI L Drug induced Diseases Boerhaave Course Leiden, 1962 (van Gorcum and Charles C Thomas)

7 WIER J A a.o Dis Chest (1956) 628

8 KERN G Schweiz med Wsch *92* (1962) 77

SOME DATA ON OUR LARYNGECTOMEES

by

A. J MOOLENAAR BIJL
Groningen, The Netherlands

A field in which Huizinga always has been interested theoretically as well as practically, is the human speech Speech as a vehicle of language, speech too as one of the functions of the organs the ear, nose and throat specialist takes care of Many of Huizinga's publications speak of this interest In all life's fenomena it has always been the mobile, the lively, the adaptable that captivate him most So he was soon gripped by these features in physiologic phonetics, which at that time in Holland were pushed forward considerably by Zwaardemaker In 1932 he made an investigation with very simple aids and in an original way of the mutual adaptation of the resonance cavities when the vowels are pronounced with open and closed mouth (N T v G *76*, 4737 (1932)) Later on he made experiments concerning the influence of consonants on following vowels (Arch néerl de Phonet Exper T XVII, 1941)

Above all he was intrigued by uncommon skills as e g ventriloquy He examined its mechanisms with the aid of X ray photographs of the larynx (Arch neerl de Phonet Exper T VI, 1931) His masterly lecture, held for the Society of Physics in Groningen in 1952 about Ventriloquists, sword swallowers and fire eaters", culminating in a find of a Shakespearian murder through the ear", brilliantly analysed, is unforgettable for all those who attended it We can only regret that Huizinga never revised it for publication

To have worked in the logopedic department of Huizinga's clinic during nearly 30 years means to have been privileged that long time to daily experiences of his wide erudition, his wit, his humanity In his patients Huizinga sees above all the human being and considers the voice as a personal attribute of that human being If ever, than this has been shown by the great many actors and singers who came to consult him because of voice complaints With their difficulties he sympathized as a matter of course

From this same human sympathizing originated the warm interest he has ever surrounded his laryngectomees with He encouraged them to achieve a new voice and created favourable conditions to this end From the very beginning Huizinga has realized the importance of the laryngectomee being revalidated immediately after reconvalescence and he recognized that this speech revalidation is the imperative duty of the clinic in which the operation took place

From this principle facts of two sorts originated the results of the speech rehabilitation and thereby the social and personal adaptation of our patients may be called good in general — and speech without a larynx has become, under

Huizinga s stimulus, an object of research about which a number of publications from the Groningen E N T Clinic have appeared

This research concerned until now mainly the physiological and physical aspects of the alaryngeal speech mechanism By means of data gathered through the years concerning the own" laryngectomees, some other more personal aspects can be elucidated now

From 1939, when Huizinga did his first laryngectomy, until the middle of 1962 this operation was performed in the clinic 105 times Of these 105 patients there were 8 with whom no speech revalidation could take place on medical indication So our further data concern 97 patients (among them 5 women) who immediately after recovery got speech treatment during their stay in the clinic, in order to master an oesophageal voice

With a later qualitative review of their speech, which usually takes place on any given moment but not earlier than 4 months after the beginning of speech treatment, the oesophageal speech could be called good" or excellent" (in dicated by marks 3 and 4) for 69 patients (71 %), moderate" or bad' (marks 2 and 1) for 28 patients (29 %) One of the 5 women belonged to the last group

Apparently the age at the time of operation and therefore at the beginning of the speech revalidation plays an important part none of the 24 patients younger than 51 was considered to have a bad" or moderate" speech, whereas 10 out of 17 in the age group between 66 and 75 were given the marks 1 or 2 (see table 1)

TABLE 1

Age at operation	Number of pat	Speech reval	Marks 3 4	Marks 1, 2	Percentages Marks 1 2
< 30	1	1	1		
31—40	4	4	4		
41—45	6	6	6		
46—50	14	13	13		
51—55	19	18	14	4	22 %
56—60	21	20	12	8	40 %
61—65	18	17	11	6	35 %
66—70	14	13	6	7	54 %
71—75	6	4	1	3	75 %
76—80	2	1	1	0	
	105	97	69	28	

A very important point for those who are too young to retire on a pension is the question whether they can carry on their profession This is not always possible, either because the work requires too much physical strain or because too high demands are made upon the voice

About this and some other points concerning the personal adaptation, we have gathered information from 40 out of the 97 patients, who represent all years of operation and all age groups except one (Table 2)

TABLE 2

Age at operation	Number of patients questioned
< 40	3
41—45	4
46—50	7
51—55	7
56—60	7
61—65	4
66—70	7
71—75	0
76—80	1
	40

As to professional work, 10 out of 40 were above pension age or had retired on a somewhat advanced pension, 15 of them were able to resume their own profession in whole, 10 did so partly, 5 had to take another job a schoolteacher became a schooladministrator, a pub-keeper became a caretaker, a baker and a taxi driver became factory workers, a navvy started a business in second hand motorcycles So the latter changed his toil for activities in which he had to talk a lot!

The time passing between the beginning of the revalidation and the moment the patient can passably manage with his new voice varies from one week to, exceptionally, over three months On an average this process takes about three weeks Correlation between duration of this period and the final result appears to be significant, as 4 out of the 11 patients whom it took over 4 weeks to come to a serviceable oesophageal voice, only reached the appreciation "moderate" or "bad", whereas the 29 patients who were able to carry on their daily conversation amply within four weeks all except one kept to their "good" or "excellent" marks

Beside the 34 patients who confine themselves to conversation within their own circle, there are 6 who speak occasionally in a larger company, e g in a board meeting or a meeting of their professional association Recently a 40 years old patient has addressed a large auditorium as chairman of a celebrating society He did this 8 months after his operation, clearly intelligibly and without any fatigue

To the question so often put by outsiders, whether the oesophageal voice is fatiguing, 21 answered "never" 19 said "now and then" (mainly after prolonged talking)

Among the conditions which make even the best oesophageal speech difficult,

irritation of trachea and bronchial tubes plays an important part 39 times a cold or a cough was mentioned as a real impediment Second comes peristalsis, that makes it impossible for 34 patients to take part in the conversation during the meals Bodily effort (e g ascending stairs) was found to impede oesophageal speech by 31 patients While lying on their back 29 cannot speak well, during the first hours after awakening speech is not fluent with 22 patients All patients agree that emotions decrease their command of the voice

Three of our patients can whistle again, two of them excellently so Analogous to buccal speech' we might call this buccal whistling" These exceptions however prove the rule that the inevitable impoverishment of phonic expression (apart from loss of speech melody) is subjectively felt as a real loss not to be able any more to sing, to hum, to groan, to laugh out

This once more underlines how much the voice is a hyper individual means of expression The obvious conclusion is that the highest possible level of oesophageal voice should be striven after with all means available In Huizinga's clinic this conclusion has always been the leading principle

Thinking of Huizinga's maxim that the clinic should pass on its knowledge, this article may be hold unpretentious evidence of my affection and admiration for Huizinga and of my gratitude for all he has taught us, intentionally and un intentionally

PURPOSIVE INATTENTION TO OLFACTORY STIMULATION

by

V E NEGUS, D Sc
Haslemere, England

I remember very clearly going to a market in a small English country town where my grandfather lived and seeing there a man who claimed to extract teeth painlessly He had a high farm waggon with steps to climb up and a chair on which the victim sat Needless to say there was a large audience of country folk

The anaesthetic was administered by a band of four or five instrumentalists, one had a brass double bass and another a very large drum When all was ready the band played softly and the patient opened his mouth, as the dentist applied his forceps the band rose to a crescendo and at the moment of extraction the double bass emitted a terrifically loud blast and the drum received a violent blow The sudden noise was ear splitting and so stimulated the victim's auditory apparatus as to make him unconscious of the pain in his dental nerve

I met several instances in the late war of individuals whose tympanic membrane had been ruptured by the blast of a nearby exploding bomb, the excruciating noise prevented them from feeling any pain

Many lovers of music prefer to shut their eyes while listening so as to concentrate on the sense of hearing It appears impossible to receive more than one stimulus to the full at one and the same time

This interference of perception in one part of the cerebrum by excessive stimulation of another area occurs, I believe, in some animals equipped with an apparently efficient olfactory apparatus, as judged by the large area of olfactory mucosa in the nasal fossa and in the frontal and sphenoidal sinuses where ethmo-turbinals have extended to increase the total area A wide air-conditioning apparatus is provided in extensive maxillo-turbinal bodies in keen scented carnivores and gives further evidence of their olfactory potentialities

An example is the Lion, whose anatomical arrangement is as good as that of others of the cat tribe known to have keen powers of scent The Lion hunts mainly by sight and even more so does the Cheetah who also is well equipped for olfaction Apparently both these animals ignore olfactory stimuli in favour of sight and probably of hearing

The Tiger, with an anatomical apparatus as good as that of the keen scented dogs and wolves, is said by some to hunt by sound and sight, but since these animals seek their prey often in dense forests and at night it seems probable that they rely greatly on olfaction

It would appear, from anatomical examination, that the Lion or Cheetah might receive embarrasing stimuli in the olfactory area in the presence of some strongly odorous matter, for instance the remains of an antelope or other animal left over

from a previous kill But if he were to make the fullest use of sight and hearing he would have to concentrate his cerebral perception on these senses and would not pay attention to that of olfaction It would be necessary to ignore the latter sense either by automatic supercedence of the optical and auditory stimuli or alternatively by training, which in all probability is the correct answer

Of Bears, the Brown, Himalayan and American Black species are known to be keen scented, but the Polar Bear, who possesses a similar anatomical structure, cannot rely on scent in his frozen Arctic habitat

Seals and Walruses, living in the Arctic, show regression of the ethmoidal turbinals which are not only reduced in area but are almost entirely surrounded by bony walls It would appear that they have lived so long in cold regions that their olfactory apparatus, no longer of practical use, has regressed, as with higher primates and Man

The Polar Bear, on the other hand, retains an elaborate ethmo turbinal system which suggests that use of the sense of smell when in relatively warm surroundings, as in an ice or snow cave and then with the requirements of recognizing food or of distinguishing between others of the species, including offspring, is still of advantage, alternatively the species, not of very ancient origin, has not lived long enough in cold regions to show regression of the olfactory receptors

The complexity of the air-conditioning maxillo turbinals, of vital importance to most carnivora for entrapping olfactory molecules gives no indication of relative olfactory activity in Arctic species, because it has been retained for the benefit of the respiratory tract and maintenance of body temperature by virtue of its power of supplying warmth and of retaining heat on expiration This applies not only to Polar Bears but also to Seals and Walruses in which the maxillo turbinals show intricate branching and subdivision thus providing a wider area than in any other animal

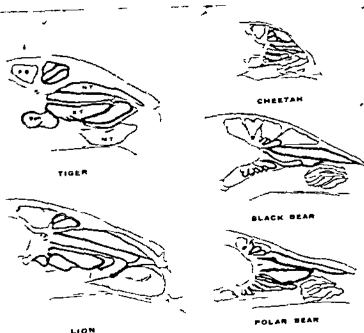
This short communication is written with the object of discussing the possible rejection of one sense in favour of another of more immediate importance, and it is I believe a problem of interest to our Colleague in whose honour it is composed I have every confidence that if he directs his alert attention to the subject he will be able to provide the correct answer

In an attempt to resolve the problem I have taken the obvious course of consulting a neurophysiologist and have been fortunate in obtaining the opinion of Doctor Barry Wyke of the Department of Physiology to which I myself am attached at the Royal College of Surgeons of England

The explanation is forthcoming and is set out in a booklet by Dr Wyke on Neurological Aspects of Hypnosis[1] from which the following quotations are taken

Extending throughout the length of the brain stem in all vertebrate animals, including Man there is a long column of interconnected cells known as the reticular system The cells of this system receive afferent connections from all the

1) *Neurological Aspects of Hypnosis* Dental and Medical Society for the Study of Hypnosis London



Drawings by Eve Negus

Similarity of structure with variation in function

In each of the five skulls, mounted in sagittal section the area of olfactory mucosa is in dark shading and is very extensive (E. T and N T) The corresponding large maxillo-turbinal (M T) is faintly shaded and so are the cavities of the frontal sinus (F S) and the sphenoid recess (Sph)

The extent of ethmoidal turbinals of the Lion corresponds closely to that of the Tiger, while the Cheetah is very slightly less

There is little difference between the structure of the Black Bear and that of the Polar Bear

E T = Ethmoidal Turbinal, N T = Naso Turbinal M T = Maxillo Turbinal, F S = Frontal Sinus Sph = Sphenoid Recess

sensory tracts ascending into the brain from the spinal cord, and from the sensory roots of the cranial nerves In turn, the reticular cells project upwards to all areas of the cerebral cortex through a series of relays in the upper part of the brain stem, in what is known as the reticulocortical projection system"

Each individual at any moment of time perceives only a tiny fraction of the mass of information being poured into his neuraxis from receptors inside, and at the surface of his body This perceptual selection is attention

It has been clearly established that incoming sensory impulses from sensory receptors can be selectively facilitated or inhibited at their point of entry into the neuraxis A considerable fraction of potential sensory experience is normally reduced or blocked before it ever reaches the brain The block occurs at the first

synapse after the sensory nerve fibres enter the spinal cord, and similarly with the visual, olfactory and acoustic systems

Attention leads to perception of small numbers of selected sensory stimuli For instance, if attention is directed to a visual stimulus, then simultaneously evoked acoustic potentials are blocked, when they reach the cells of the cochlear nuclei, by discharge of inhibitory impulses from the reticular system "

Sensory perception is dependent upon attention which, by shifting from moment to moment as it does in normal life, selects limited information from the environment to the exclusion of other stimuli

This explanation fits the problem to which this communication is directed, it depends, not on hypothesis or speculation, but on a very large amount of experimental investigation by many research workers

A SYSTEM FOR ILLUMINATION OF THE MIDDLE EAR THROUGH THE EUSTACHIAN TUBE IN TYMPANOSCOPY AND LABYRINTHOSCOPY

(Preliminary Communication)

C O NYLÉN and B G KARLSSON *

Uppsala, Sweden

During *otoscopy* observations are made through the external auditory meatus with varying illumination However, no experiments seem to have been made where the middle ear is locally illuminated from the Eustachian tube

During 1962, the authors carried out experiments on three patients who had recently died, one at forty years of age and the other two at eighty years of age, and on five specimens from patients who had recently undergone postmortem, all adults of a high age group

Technique A homogeneous light guide of Lucite was used Its total length was 20 cm of which 10 cm had a diameter of 2 mm and the remainder only 1 mm With the latter part ahead, it was passed into the middle ear through a tubal catheter, placed in the choanal orifice of the Eustachian tube The light was conducted from a spotlight of 30 W through an optic light guide of flexible glass fibres about 1 m in length and 5 mm in diameter (American Optical Company), connected to the free wider end of the Lucite rod (Fig 1) This arrangement

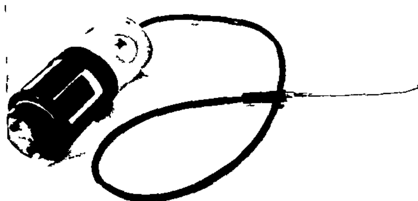


Fig 1 Instrument for local illumination of the middle ear The 1 mm wide top of the Lucite light guide is seen emerging from the tubal catheter

* *From the Department of Otolaryngology and the Gustaf Werner Institute, University of Uppsala*

permitted suitable flexibility and thermal isolation between the light source and the heat sensitive Lucite

In every case except one (sometimes after repeated examination), the probe successfully passed through the Eustachian tube However, there was some difficulty in passing the isthmus The authors can confirm reports from other workers (see the references) concerning the large variations in width and length of the Eustachian tube

When the Lucite rod had been pressed through the isthmus of the tube, after a further 1—1 5 cm, it entered the middle ear and the tympanum was illuminated by the light emanating from the rounded top of the Lucite rod In the first three cases, the light through the tympanic membrane was slightly rose coloured, probably due to blood still present in the vessels In the specimens treated with formalin, the faint colour was almost or completely absent

Subsequently the Lucite rod could be introduced 1 cm further into the middle ear and against the mastoid antrum In this last position the light seen through the tympanum was reduced

Our attempt at local illumination of the middle ear through the Eustachian tube was made to facilitate the possible study of the internal ear through the round and oval windows (after removal of the stapes) by *labyrinthoscopy* and by the collection of specimens with a capillary tube from the labyrinth under visual control The instrument described may also be of some use in clinical *tympanoscopy* as a supplement to conventional optical systems for observations of the middle ear

REFERENCES

ASCHAN G Acta Oto-Laryng, Stockh Suppl *69* 1948
— Upsala Lak foren Forh *57* 1 (1952)

BAST Th H and ANSON B J The Temporal Bone and the Ear Ch C THOMAS, Spring field, Ill., U.S.A, 1949

PORTMANN G RETROUVEY H and LACHAPELE, R Rev Laryng 44 849 (1923)

RAUBER and KOPSCH Vol 3 424 Leipzig 1939

SCOTT BROWN W T Diseases of the Ear, Nose and Throat Vol II, p 14 Butterworth and Co London, 1952

SPIELBERG W Arch Otolar *5* 334 (1927)

WELIN S Acta Radiol *28* 95 (1947)
— Nordisk Larobok i Oto-Rhino-Laryngologi 17 1958 Munksgaard Copenhagen

ZÖLLNER F Anatomie Pathol u Klinik d Ohrtrompete Springer Verlag Berlin 1942

CYTOLOGIC EXAMINATIONS OF SPUTUM IN PATIENTS WITH UNCOMPLICATED INFLUENZA

by

N G M ORIE, A LÖWENBERG, F J KEUNING *

Groningen, The Netherlands

Part of the cases of pneumonia[1], complicating influenza and part of the (late) fatal outcomes in these patients are readily explained by preexistent lesions mainly of the heart and the lungs[2]

Influenza occurring in these patients is often complicated by a bacterial inflammation, in which as a rule H influenzae and pneumococci are found[3] The bacterial superinfections and the course of the disease are in no way different from that in spontaneous" pneumonic processes, occurring in patients with chronic bronchopulmonary (or cardiac) disease

In the other, mainly severe (often early!) fatal cases we are usually dealing with young vigorous people The bacterial flora in these cases in the latest epidemics consisted mainly of staphylococci[3] Mulder has pointed out that a source of virulent staphylococci in these cases usually can be demonstrated

Whereas in the first group (which caused in our country in the 1957 and 1959 epidemics ± 70 % of the complicated cases of influenza[1] and a similar" although somewhat lower percentage of the fatal cases) the complications seem fairly well explained, this is by no means true in the second group

One may wonder whether the conditions in which these staphylococcic complications occur are not provided by the presence of serious lesions of the bronchial epithelium not found in the majorities of the uncomplicated cases The serious lesions of the mucous membrane could be the expression of a lowered resistance

This resistance is, in mainly infectious diseases — tuberculosis[14] — definitely related to age and sex The young girl (Mimi from La Boheme!) of old is the prototype of the serious case of tuberculosis

Thus the preference of this form of fatal infections for the young adult and specially for the female could be understood

But even then is this vulnerable group only a single individual falls a victim Possibly a more detailed knowledge about incidence and severity of cytopathic

* *From the Department of the Medical Director (Dr P Bolt), the Pulmonary Division (Prof Dr N G M Orie) of the Department of Internal Medecine (formerly Prof Dr F S P v Buchem) and from the Laboratory of Histology (Prof Dr F J Keuning) State University, Groningen*

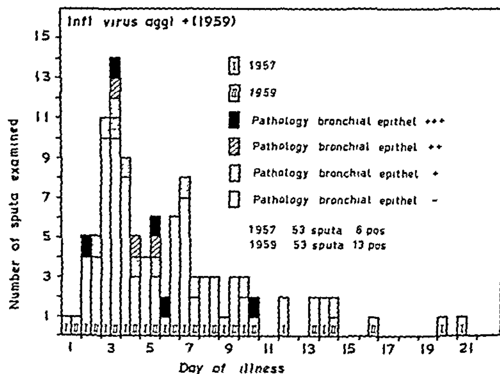
** *With technical assistence of Miss A. Kikstra*

lesions in the bronchial tree could provide us with a more exact knowledge of the conditions determining an unfavourable course of an influenza infection

Cytopathic lesions in influenza

Cytopathology is a characteristic of viral infections in general, and particularly of influenza The cytopathological effect of influenza virus in tissue cultures has been experimentally assessed

The injuries of the respiratory epithelium in ferrets and mice are also beyond doubt Stripping" occurs after 2 × 24 hours, regeneration starts a few days later, and is completed 10—14 days afterwards



Lastly it is certain that in some cases of human influenza considerable destruction of the respiratory epithelium takes place

It is true that destruction of the mucous membrane is found in many post mortem cases, but here naturally we are dealing with a selection, in which the epithelium destruction may have been the cause of the fatal outcome

Comparatively little is known about the frequency of the occurrence of cytopathic phenomena in epidemic and sporadic cases of influenza[5] An investigation was therefore set up during the influenza epidemics of 1957 and 1959

Material and methods

Sputum smears were examined, once or several times in the course of the disease in members of the nursing staff of the Academic Hospital, during the top of the epidemic of 1957 and in 1959. They were uncomplicated cases, mostly in young female patients.

In the first series only some of the cases were virologically confirmed, but in our opinion there cannot be any doubt about the diagnosis in view of the epidemic course.

The series of cases from 1959 were confirmed serologically.

The results of these cytologic examinations were compared with those in similar patients in which influenza had been excluded, by serology as well as on epidemic grounds.

In 33 such patients sputum smears with abnormal cells were not seen.

The staining was done according to Papanicolaou[6], the following criteria were used for the assessment of cellular damage:

1) Changes of columnar epithelial cells
   a. decrease of cell size and loss of cilia
   b. progressive loss of typical cell shape
   c. progressive pycnosis of cell nucleus

2) Appearance of abnormal small round cells with pycnotic round nucleus and little or no cytoplasm. The origin of which is not clear.

Results

Findings in cases of influenza without complications.

Fig. I shows the results of sputum cytology in all members of the nursing staff fallen ill probably with influenza between 19/9 and 28/11 1957 (40 patients), and those fallen ill in 1959 and where the diagnosis influenza A or B could be confirmed serologically (43 patients).

The results show, as could be expected, that the majority of the positive findings (15/19) were obtained during the first week of the disease.

There was no important difference between the epidemics of 1957 and 1959.

Although the possibility does exist that a positive result is dependant on the sickness day on which the sputum sample was taken, the distribution of the positive results during the first week of the disease does not support this view.

In some cases of course early samples were positive, those taken afterwards negative. In one case findings were negative on the second, positive on the third day of the disease.

Although the distribution of the positive results points to the conclusion that there is no definite preference for a particular day of the illness, positive findings hardly can be expected after the 8th or 10th day. (Most patients did not produce any sputum in later stadia.)

No distinct difference could be assessed in this small series between the incidence of cytopathic effects in influenza A and B

Only in three samples of sputum mainly mononuclear cells were found The small quantity of predominantly mucous sputum contained in the majority of cases polynuclear cells

No clear answer to the interesting age sexproblem was possible in this group the majority of the patients being young women (nurses), but in this group of patients the epithelial damages seem to be fairly limited

SUMMARY

The incidence and degree of cytopathic changes of the respiratory epithelium as found in the sputum of 83 patients with uncomplicated influenza is described

Clearcut abnormal cells could be demonstrated in 20 % of the cases

Most patients were young women.

The absence of haemorrhagic sputum the small amount of sputum produced and the absence of cytopathic changes in the majority of the cases of uncomplicated influenza suggests that in this type of patients the lesions of the respiratory epithelium are usually slight or nonexistent

No difference between the 1957 and the 1959 epidemics could be established The findings in influenza A and B were identical

It is self evident that we who have had the benefit of Prof Huizinga's personality and skill (when not abroad) for so many years are particularly anxious to join our compliments and well wishes to those of so many distinguished scientists

REFERENCES

1) ISRAELS A. A and v d WAL A Bronchiectasis Bronchitis An International Symposion Van Gorcum Assen (1961)

2) VAN GEER I and MULDER J De sterfte aan Aziatische Influenza in 1957 Influenza p 42 Boerhaave Cursussen Leiden (1958)

POLAK, M F Influenzasterfte in de herfst van 1957 Influenza p 46 Boerhaave Cursussen Leiden (1958)

3) GOSLINGS W R O Bacteriele complicaties van influenza en hun prophylaxe en behandeling. Influenza, p 48 Boerhaave Cursussen Leiden (1958)

4) GRIEP W A De tuberculine reactie Royal Dutch Assoc for Tuberc (1957)

5) HERS I F Ph The histopathology of the respiratory tract in human influenza Leiden 1955 (Thesis)

6) PAPANICOLAOU G N Atlas of exfoliative cytology Harvard Univ Press Cambridge Mass (1954)

DIFFERENTIAL DIAGNOSIS OF SHADOWS IN THE LUNG FIELDS

by

F C ORMEROD

London, England

It is a pleasure to contribute an article as a compliment to Eelco Huizinga He has played such a prominent part in Otolaryngology for the past forty or more years — an original member of the Collegium President of the International Congress in Amsterdam, multilingual and a self appointed interpreter, an orator and not least a most respected otolaryngologist whose work on the internal ear and, perhaps slightly unexpectedly, on the bronchus have enriched our specialty and brought him much fame By his kindliness and his friendliness and his wit, he has endeared himself to all who know him and this includes almost everyone who practises otolaryngology, and none can leave his presence without feeling happier and more at peace with the world

Huizinga has written so much about lesions in the bronchial tree and the lungs that a comparative record of a variety of conditions encountered in this field during the course of bronchoscopic service of many years may not be considered out of place

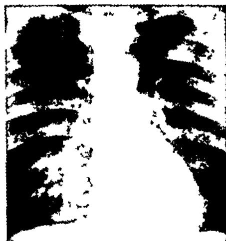
A series of eighteen radiographs has been selected six each of the upper middle and lower zones of the lungs, from pictures of opaque appearances in the lung fields, the diagnosis of which had been established by endoscopic inspection In selecting these radiographs, those which represented simple inflammatory disorders of the broncho-pulmonary system have been regretfully omitted as their inclusion would have trebled the length of this communication

Plate A.

In the upper zone a primary tuberculous focus as described by Assmann, circumscribed and regular in outline and independent of bronchial anatomy can be confidently indentified by calcareous areas In the next case a metastatic seminoma also shows a smooth, spheroidal area free in the parenchyma of the lung Diagnosis might have been difficult but for a history of orchidectomy three years previously with available histological description A similarly placed though larger shadow in the next radiograph shows the irregular margin and the variable density of a primary carcinoma without as yet any atelectasis due to bronchial occlusion The fourth radiograph demonstrates a total atelectasis of the right upper lobe which was caused by the presence of an enlarged tuberculous lymph node on the eparterial bronchus the occlusion of the bronchus being easily recognizable endoscopically The fifth radiograph shows a similar picture of a collapsed upper lobe but this was due to a [illegible] of the upper lobe bronchus and presenting in the main bronchus The [illegible] of this series is of a carcinoma of the upper lobe which [illegible] the upper lobe rather than causing atelectasis



1 Assmann's tuberculous focus



2 Seminoma (metastatic)



3 Carcinoma (primary)



4 Atelectasis from pressure of tuberculous lymphnode on upper lobe bronchus



5 Atelectasis due to adenoma



6 Atelectasis due to carcinoma (primary)
Compared with the two previous cases the lower margin is uneven and not smooth and regular as seen in the atelectatic cases

Plate B

In the midzone an irregular shadow is extending from the hilum into the lung parenchyma without causing any pulmonary atelectasis A lateral view showed that the main bronchus was still patent, but endoscopy revealed narrowing of its lumen Subsequent progress showed the shadow to represent a hilar carcinoma The second radiograph shows a similar though smaller shadow in the left hilar region This was caused by metastatic deposits of tumour cells in the lymph nodes of the mediastinum, a lymph borne extension of an unsuccessfully treated laryngeal carcinoma The third of this series shows a circumscribed shadow, with a smooth outline, free in the parenchyma This proved to be a primary bronchial carcinoma which originally presented with a metastatic deposit on the aryepiglottic fold, and later spread to almost every tissue in the body The fourth, a very large shadow in the right lung field consisted of a mass of fibrous tissue, which was described as a fibroma but histologically with some suspicion of sarcomatous change The next one with an opacity in the same area, represented atelectasis of the pectoral segment of the right upper lobe, which was caused by occlusion seen by endoscopy, of the appropriate bronchus by the pressure of a tuberculous lymph node On caseation of the node, the pressure was released and the lung segment reaerated The final radiograph of the series represented a condition in a lady of 74 years with a large spheroidal cavity and much opacity The diagnosis was facilitated by the discovery (in 1931) of free carcinoma cells in aspirated sputum

Plate C

The first radiograph shows a roughly spheroidal shadow with an irregular margin, and with a variable degree of opacity, with some additional shadowing of the lung Endoscopy showed only inflammatory thickening of the bronchial mucosa, but a Wassermann reaction and immediate and complete response to specific treatment demonstrated that the condition was a gumma The next radiograph shows a primary carcinoma of the posterior segment of the lower lobe, with the typical irregular surface and variable density The third represents two blood borne metastases, from a carcinoma of the larynx, situated in the right lung They show the smooth outline, the more spheroidal shape and the more uniform density of the secondary — or cannon ball — carcinoma The fourth and fifth radiographs also demonstrate rounded opacities with smooth surfaces and uniform densities They are not secondary carcinomata the first being a hiatus hernia and the second a hamartoma of the median basal segment of the lower lobe The sixth radiograph shows a large smooth opacity continuous with the left ventricular region of the heart It was caused by a somewhat ectopic thymoma — diagnosed by transthoracic resection

These radiographs illustrate a few of the wide range of pathological conditions in the lung fields the identification of which is very much facilitated if not always finally determined by bronchoscopic measures

LE SYSTÈME VAGO SYMPATHIQUE ET L ÉQUILIBRE TENSIONNEL DU LABYRINTHE

par

GEORGES PORTMANN

Bordeaux France

La connaissance des rapports entre le systeme vago-sympathique et la pression endolabyrinthique est la base meme de la pathogenie des vertiges et commande les traitements tant médicaux que chirurgicaux

A un systeme arteriel terminal auriculaire particulierement developpe est adjoint un système sympathique périvasculaire très riche d origine carotidienne interne et vertébrale pour le labyrinthe postérieur

Relations vago sympathiques et labyrinthe anterieur

Les travaux de François Frank Lannois Huizinga Terracol et mes propres recherches sur la sympathicectomie periartérielle au niveau de la carotide interne ou de la carotide primitive sont significatifs Cette intervention prouve une augmentation de l audition temporaire d une duree de dix à quinze jours dûe à l augmentation de l apport vasculaire Par contre pas d amelioration des bourdonnements sauf dans un cas de Lannois sur un malade de Jaboulay

L action sur le sympathique cervical et pericarotidien a pour resultat l apparition de phenomènes vaso-moteurs au niveau des differentes parties de l oreille

— Vaso-constriction et ischémie s il y a excitation

— Vaso-dilatation et hyperemie s il y a section

Relations vago sympathiques et labyrinthe posterieur

Le sympathique cervical et le pericarotidien ont une action vaso-motrice indiscutable sur l appareil vestibulaire

La section du sympathique cervical ou la sympathicectomie péricarotidienne entraine une vaso-dilatation labyrinthique

Cette vaso-dilatation se manifeste au point de vue fonctionnel toujours par de l hyperexcitabilité avec le plus souvent nystagmus battant du coté opposé

Action pharmaco-dynamique de certaines substances

Avec le Professeur J Despons nous avons experimenté sur les chiens quatre groupes de substances pharmaco-dynamiques opposees

— sympathicomimétique

— sympathicolytique

— parasympathicomimétique

— parasympathicolytique

Nous avons pu arriver aux conclusions suivantes Dans leur ensemble les médicaments du systeme organo-vegetatif ont provoque les reactions generales specifiques qui leur sont classiquement reconnues

Les medicaments ayant une action directe sur le sympathique semble provoquer une reaction vestibulaire constante les excitants du sympathique determinent de l'hyperexcitabilite les inhibiteurs du sympathique de l'hypoexcitabilite

Maisonnobe confirme chez l homme des resultats obtenus chez le chien Il en est de meme de Eelco Huizinga

Nous avons egalement essaye de determiner les relations pouvant exister entre les phenomenes circulatoires generaux et la circulation labyrinthique Soumettant les sujets a des bains chauds (38°) on observe de maniere generale une hyper excitabilite vestibulaire qu il est difficile d attribuer soit a une anemie en cephalique soit a une hypotension labyrinthique

Ces diverses constatations ont permis de degager un syndrome clinique des spasmes de l oreille interne

1° Bourdonnements vertiges
2 Surdite
3° Hyperexcitabilite vestibulaire
4 Hypertonie sympathique

Ces symptomes ont toujours ete suffisamments concordants dans les divers examens que j ai pratiques pour constituer un syndrome constant avec les seules modalites dues au territoire particulier soumis a la vaso-constriction labyrinthe anterieur labyrinthe posterieur ou les deux a la fois

A cote de ce *syndrome de resistance ou d hypertonicite* arterielle du labyrinthe nous devons placer le *syndrome d hypotonie ou de laxite* caracterise par

— Bourdonnements
— Surdite

mais avec

— Hypoexcitabilite vestibulaire
— Hypotonie sylpathotique

Ces deux syndromes peuvent d ailleurs alterner chez le meme malade

Suivant les individus on observe en effet des résultats variables par excitation du systeme vegetatif sous l influence de causes diverses Mais si le plus souvent on se trouve en presence tantot d un syndrome d hypertonie ou tantot d un syndrome d hypotonie vago-sympathique qui nous l avons vu permettent de diviser classiquement les individus en vagotoniques et sympath cotoniques on rencontre en clinique assez frequemment des sujets chez lesquels le desequilibre parait continue par une hyperexcitabilité globale du systeme nerveux vegetatif C est cet etat part culier que les auteurs ont désigne sous les noms divers de ,neurotonie (Gu llaume) desequilibre total de l ensemble du systeme (Laignel Lavastine) dyston e vegetative (Sicard) amphoton e (Daniellopolu)

Les causes de ces desordres neuro-végétatifs essentiels sont multiples méca niques endocriennes toxiques allergiques ou psychiques et l on comprend ainsi

l'importance de règles d'hygiène de diététique et de psychothérapie La thérapeutique médicale correspondra à l'étiologie de ces troubles

La chirurgie du sympathique donne des résultats certains, mais éphémères comme l'*expérimentation* pouvait le faire prévoir et la chirurgie du vertige labyrinthique s'adressera surtout à *l'hypertension labyrinthique du syndrome de laxité vestibulaire*

Les résultats de ces recherches physiologiques démontrent la nécessité de l'intégrité de l'organe endolymphatique pour le bon fonctionnement de l'oreille interne

Quelle sera donc la place du sac endolymphatique dans la physiologie de l'oreille interne

Il résorbe l'endolymphe tout au moins pour une certaine partie surtout les grosses molécules (Guild Altmann Weltner)

Il sert par sa situation, sa morphologie de tambour physiologique Il est en effet la seule partie extra osseuse du labyrinthe membraneux Ses variations de volume ne retentiront donc pas sur les autres éléments contenus dans la capsule otique

La nécessité d'un équilibre de tension osmotique et onchotique l'obligation pour les éléments sensoriels de l'oreille interne de recevoir un apport anabolique permanent d'éliminer les produits catabolisés l'importance du système nerveux organo-végétatif la protection (nécessaire pour le fonctionnement de l'oreille interne) du système capillaire local contre les troubles généraux (*d'origine humorale endocrinienne allergique ou phénomène de Reilly*) obligent à penser qu'une emprise efférente couvre cette physiologie Le rôle du matelas aréolaire que j'ai décrit en 1921 au niveau de ce que Guild a appelé la pars intermedia reste à élucider

Il existe par ailleurs un fait moins connu souligné par Secrétan à savoir la présence de phagocytes dans la lumière du sac Ils pourraient avoir une action sur les métabolites de rebut

La constance de l'organe endolymphatique dans les diverses espèces, sa structure biologique les résultats *que nous avons obtenus dans le traitement du syndrome* de Laxité par l'ouverture décompressive du sac endolymphatique posent à nouveau *l'importance de cet organe dans la physiologie de l'oreille interne*

STROBOSCOPY IN LARYNGEAL KERATOSIS AND ITS CANCERIZATION

by

ANTONIN PŘECECHTĚL *

Prague, Czechoslovakia

For the last ten years (1949—1958), at the ORL clinic of the Faculty of General Medicine at Prague, 74 keratoses of the larynx have been stated, occuring between the III and VIII decade (Tab I)

Table I

Distribution of laryngeal keratoses found during 10 years according to their occurence in the decades

Decade	III	IV	V	VI	VII	VIII	Total
Number of patients	2	9	13	31	13	6	74

The largest number of keratosis occured in the VI decade (41,8 %)
Table II demonstrates the cancerized keratoses according to the age groups

Table II

Age of cancerized praecanceroses of the larynx

Decade	III	IV	V	VI	VII	VIII	Total
Number of patients	—	3	11	12	3	4	33
% of the total of keratoses from tab I	—	33,3 %	84 6 %	38 7 %	23 1 %	66 6 %	44,5 %

Table II shows that the most praecanceroses canceroze in the V^th decade (84 6 %), thus in this decade there is the greatest trend to the cancerization of keratoses In the VIth decade however the number of keratoses was largest (Tab I), but the trend to cancer formation is already much lower (38,7 %)

The time development of praecancerosis (keratosis) of the vocal cords up to cancerization was determined from the duration of hoarseness existing before the establishment of the tumour This symptom was stated in 30 patients (Tab III)

* *From the ORL laboratory of the Academy of Sciences CSSR and the ORL clinic Charles University Prague*

Table III

Duration of praecancerosis before cancerization (30 patients)

Time of existence of the precancer before cancerization	Months		Years				
	2	6	1	2	3	5	7
Number of patients	5	11	5	4	2	2	1

Table III demonstrates that in more than a half of the cases (53,3 %) cancerization set in within six months after appearance of hoarseness, in 70 % within one year, in 90 % within three years, in the rest within 7 years

According to Tab I—III thus keratoses in the V and VI decade and the first year of development are most dangerous, as for their great incidence, as for their frequent cancerization In this age and time of development of hoarseness it is therefore necessary to take care of the dispensarisation, evidence and treatment of the patients

Adequate therapy naturally requires a precise and timely diagnosis as of the precancer as of the onset of malignant changes

The most reliable diagnosis of these structures can be naturally made by biopsy, but this method has also its difficulties and limits In extensive praecancerosis samples must not contain cancerized material, also histologic changes are not always unambiguous, and repeated excisions deteriorate the functional restitution, though the tumour may heal up well Therefore other diagnostic tools are favoured, sparing the functionally important tissues of the vibrating system, the ligament and muscle of the vocal cords

In a series of more than 20 cases of praecanceroses and initial cancer we examined the vibration of the affected vocal cord by stroboscopy and it was found that

1) Moderately developed keratosis of the vocal cord, especially flat pachydermia (pachydermia plana) does not restrict vibration
2) Verrucous hyperkeratosis of the vocal cord restricts its vibration to a certain extent usually greatly, or at all
3) In cancerization there is a total or almost total blockade of vibration

Therapy of hyperkeratoses Demarcated hyperkeratoses of the vocal cords are treated at our clinic mostly by decortication, changes of greater extent by radiation After restitution of the hyperkeratosis and gaining of normal anatomic state of structures restitution of blockaded vibration was found too In deeper scarred changes and deformation of the vocal cords there may be partial or total suppression of vibration even after healing of the keratosis

Initial carcinoma of the vocal cord is treated by radiation or chordectomy Quite exceptionally the carcinoma may be liquidated by probatory excision (2 cases)

After radiotherapy of the cancer the not vibrating cord may begin to

vibrate normally If scarring, deformation or bad closure of glottis occurs, the cord must not vibrate even after proper healing of tumour

According to our experience normal vibration testifies against malignity Abundant restitution of vibration in cancer verifies healing

If there is no vibration after radiation therapy of cancer, that must not mean that the tumour was not healed, but only scarring of the vibrating structures to a greater extent In such cases it is necessary for the statement of the real situation to observe the patient for a longer period or, in case of uncertainty, to carry out biopsy

Chordectomy usually suppress vibration Decortication and probatory excision normally does not deteriorate the vibration of the vocal cord Only quite exceptionally, as was mentioned above, probatory excision may remove the whole tumour and vibration may be preserved In such cases it is necessary to examine the patient by laryngoscopy and stroboscopy for a long period

REFERENCES

ANTONIN PŘECECHTEL, JIŘI ŽABKA PAVEL ŠKEŘIK Prekancerózy horních dýchacích cest a hrtanu Acta universitatis Carolinae Praga Supplementum *10* 1960

ANTONIN PŘECECHTĚL La stroboscopie dans le diagnostic precoce du cancer de la corde vocale Archivio Italiano d ORL Vol LXXII 3 1961

THE CASE FOR RADIOPAQUES IN SINUS ROENTGENOGRAPHY

by

ARTHUR W PROETZ

St Louis, Mo, U S A

For the past thirty years the roentgenography of the nasal sinuses has been more or less a standard procedure, with only minor variations between clinics and countries

This is somewhat surprising in view of the shortcomings of certain better known positions and their diagnostic limitations The posterior anterior 55° (Waters) position is in common use notwithstanding the fact that, in dropping the petrous pyramid to a level where it does not obscure the maxillary sinus, the floor of the sinus has been tilted to a point where it is itself obscured by the dental roots and the alveolar ridges The density of these structures is sufficient to hide completely even one or two ml of a fluid radiopaque Soft tissues, of course, are likewise hidden

Lateral views of the sphenoid cavity also involve six or seven superimposed, variable bony structures which, while they may be distinguishable by an expert in a normal nose, are confusing to anyone in the presence of hyperplasia, erosion or tumor masses

Since 1925, only shortly after their discovery, my interest has been focused upon the behavior of fluid radiopaques introduced simultaneously into all the sinuses by the intermittent suction technique, displacement

In the beginning this procedure was employed only in *difficult* cases, but the results were so revealing and the technique so simple that it soon became routine *with me and the study* material is endless

Most of the published illustrations of sinus films, in the current literature, show *no contrast* media *and too frequently* one could wish *that they did*, especially when the reproduction is less than perfect

What *follows is the briefest statement of personal conclusions* after four decades of observation

Diluted radiopaques are preferable to the undiluted product (we use Lipiodol, one part to three of olive oil) first, because overlapping shadows such as occur between ethmoids and sphenoids are more easily distinguished, second, because the viscosity is ideal for the purpose, third, because a standard viscosity is required for an accurate estimate of emptying time, and, fourth, because the cost per patient becomes negligible Eight ml of oil is used

Partial filling is preferable not because complete filling is possible only when the ostium is at the exact top of the cavi t because, by shifting the position of the head the oil can be gotten out y when it is desired to view a given area without it

The horizontal beam is used, for obvious reasons, and three positions of the head are standard practice the direct postero-anterior view with the base-line horizontal (0° Granger), the lateral view, a 90° rotation from the last with the base line still horizontal, and the sub-mento-vertical view, with the base-line vertical These are namely the front elevation, the side elevation and the ground plan" of the engineer and are similarly informative and mensurable

With radiopaques and accurate standard positions any anatomical departure from the normal such as a pneumatized pterigoid process or sphenoid wing becomes instantly apparent

Radiopaques plainly distinguish between thickenings of the types of sinus mucosa whose outlines do not follow bone contours (as in allergic and similar edemas, inflammations and other reversible conditions which respond to treatment) and those which do follow the bone contours (old hyperplasias which are not reversible and require surgery, if anything)

Perhaps the most rewarding feature of displacement with radiopaques, and one which can be accomplished by no other means, is the demonstration of the extent to which a given cell or sinus can drain itself without help, and then of supplying help where it is needed Filling by displacement is dependent upon four conditions 1) The ostium must be reached by the fluid, in fact submerged in it, 2) The negative pressure must reach the ostium, 3) The ostium must be anywhere beside the exact bottom of the sinus, and 4) The ostium must be patent

The first three are under the control of the operator The fourth tells him that any sinus which fails to fill (or to empty) has ostium trouble, which is after all, the point of the investigation

Any fear of spreading droplets of oil into the tissues is unfounded since the pressure used is a negative pressure If this is kept to −150 mg Hg as prescribed there will be no injury

TABELLE I Einzeitige Divertikelresektion <u>ohne</u> Durchtrennung des M. cricopharyngeus

(Fortsetzung)

FALL	OPERATIONS-JAHR	PRAEOPERATIVE BESCHWERDEN	PRAEOP. ROENTGEN	JAHRE SEIT OP	HEUTIGES ROENTGEN	HEUTIGE BESCHWERDEN
13 K.H. ♂ 56 J. 220/54	1954	Regurgitieren Zaher Speichel		8		keine
14 K.O. ♂ 56 J. 652/53	1953	Druckgefühl Steckenbleiben Reizhusten Würgen		9		keine Recidivoperation !
15 L.F. ♀ 53 J. 72/60	1960	Druckgefühl Regurgitieren Gewichtsabnahme		2		keine vorübergehende Recurrensparese links
16 M.H. ♂ 61 J. 154/59	1959	Reizhusten Regurgitieren		3		Luftschlucken und Aufstossen selten belegte Stimme laryngoskop. oB. + Hiatushernie
17 M.F. ♂ 60 J. 370/58	1958	Schleimspucken Schluckbeschwerden Regurgitieren		4	fehlt	keine Recidivoperation !
18 M.E. ♂ 66 J. 370/56	1956	Regurgitieren Reizhusten Divertikel ausdrückbar		6	fehlt	lageabh. Regurgitieren
19 F.M. ♂ 37 J. 269/58	1958	Schluckbeschwerden Glucksen	fehlt	4		keine
20 R.G. ♂ 61 J. 606/60	1960	Schluckbeschwerden Regurgitieren			fehlt	keine
21 S.F. ♂ 51 J. 1026/59	1959	Druckgefuhl Steckenbleiben		3		keine
22 St.F. ♂ 63 J. 1010/53	1953	Steckenbleiben Regurgitieren		9		keine selten belegte Stimme laryngoskop. oB.
23 W.H. ♂ 47 J. 848/55	1955	Steckenbleiben Reizhusten Regurgitieren		7		keine

subjektiv beschwerdefreien Recidivfalle, verglichen mit den praeoperativen Bildern, Oesophaguseingange, die bedeutend tiefer liegen und weiter sind

Diskussion

Die grosse Zahl der Hypothesen, welche die Entstehungsweise des Zenker'schen Divertikels zu erklaren suchen, zeigt, wieviele aetiologische Unklarheiten noch bestehen KODICEK und CREAMER haben die [illegible]ische Theorie, wonach ein

Ueberdruck im Hypopharynx fur die Divertikelbildung verantwortlich gemacht wird, durch vergleichende Druckmessungen am Gesunden und Kranken zu uberlegen versucht Die Theorie der „neuromuskularen Inkoordination" als Ursache der Dysphagie (NEGUS) wird durch die Beobachtungen von ASHERSON in Frage gestellt KIRCHNER's Versuche deuten daraufhin, dass vor allem vegetativ Stigmatisierte, besonders sympathikotone Individuen zu Divertikelbildung neigen Allem Anschein nach bedarf es zur Ausbildung eines Zenker'schen Divertikels eines

TABELLE II Recidivfalle nach Divertikelresektion ohne Cricopharyngeusdurchtrennung

FALL	OPERATIONS JAHR	PRAEOPERATIVE BESCHWERDEN	PRAEOP ROENTGEN	JAHRE SEIT OP	HEUTIGES ROENTGEN	HEUTIGE BESCHWERDEN
24 B O ♂ 43 J. 1251/57	1957	Schluckbeschwerden Verschlucken Regurgitieren		3		keine RECIDIV
25 F J. ♂ 68 j 80/54	1954	Wurgen Regurgitieren Gewichtsabnahme		8		keine vorubergeh Recurrensparese RECIDIV
26 G P. ♂ 52 j 323/59 1175/59	1959	Regurgitieren Schluckbeschwerden		3		Schlucken fester Speisen erschwert, Reizhusten, Regurgitieren Fremdkorpergefuhl, belegte Stimme Recurrensparese re 2. RECIDIV + Hiatushernie
27 G K. ♂ 51 j 528/58	1958	Steckenbleiben Regurgitieren Reizhusten Gewichtsabnahme		4		keine RECIDIV
28 H O. ♂ 51 j 117/56 534/56	1956	Steckenbleiben Regurgitieren bei Eintritt Schluckunmöglichkeit		6		seit 1 Jahr: Schluckbeschwerden, lageabh Reizhusten und Regurgitieren RECIDIV
29 St H. ♂ 66 j 159/58	1958	Steckenbleiben Reizhusten Regurgitieren		4		Steckenbleiben Reizhusten Gluckspgerausch RECIDIV

Zusammentreffens mehrerer *disponierender Faktoren,* die zudem uber lange Zeit wirken mussen

1 Umschriebene Wandschwache in der Hypopharynxhinterwand (Killian'sches Dreieck)

2 Ueberdruck im Hypopharynx
   a) infolge Spasmus des m cricopharyngeus,
   b) infolge mangelhafter Erschlaffung dieses Muskels.

3 gewisse psychische Faktoren

Die *Symptomatologie und die Diagnose* des Zenker'schen Divertikels darf als bekannt bezeichnet werden

Die *Behandlung* besteht in der meist einzeitig vorgenommenen Resektion des Divertikelsackes von aussen (Bronchologen empfehlen neuerdings die endoskopische Schwellendurchtrennung nach DOHLMAN). Die Durchtrennung des m. cricopharyngeus wird von AUBIN, ESCHER, LAHEY und SEIFFERT gefordert. Die definitiven Ergebnisse der Divertikeloperation können erst nach einer langen

TABELLE III Einzeitige Divertikelresektion mit Durchtrennung des M. cricopharyngeus

FALL	OPERATIONS-JAHR	PRAEOPERATIVE BESCHWERDEN	PRAEOP ROENTGEN	JAHRE SEIT OP	HEUTIGES ROENTGEN	HEUTIGE BESCHWERDEN
30 dR.Ch. ♀ 61 J 415/50	1950	Regurgitieren		12		keine
31 B.P. ♂ 49 J. 571/51	1951	Schluck- und Atem-beschwerden Heiserkeit Regurgitieren		11		keine vorübergehende Recurrensparese
32 F.J. ♂ 45 J. 785/52	1952	Stossen im Hals Steckenbleiben Regurgitieren		10		keine bis 1961 jetzt: selten Steckenbleiben
33 H P. ♂ 56 J. 1160/49	1949	Reizhusten Gewichtsabnahme		13		lageabh Husten und Fremdkörpergefühl + Hiatushernie
34 K.L. ♀ 60 J. 452/50	1950	Steckenbleiben Regurgitieren		12		keine belegte Stimme laryngoskop. oB.
35 M W. ♂ 40 J 373/51	1951	Würgegefühl Reizhusten Regurgitieren		11		keine
36 W J ♂ 39 J. 623/52	1952	Fremdkörpergefühl Schluckbeschwerden Regurgitieren		10		keine
37 W W. ♂ 34 J 600/50	1950	Steckenbleiben Regurgitieren Gurren Gewichtsabnahme		12		keine
38 M W. ♂ 40 J 544/50	1950	Steckenbleiben Regurgitieren		12		keine RECIDIV

postoperativen Periode und an Hand von röntgenologischen Nachuntersuchungen beurteilt werden. In 22 Fällen, die von COVEN und ESCHER mit Cricopharyngeusdurchtrennung operiert wurden, ergibt die Nachuntersuchung bei allen Patienten subjektive Beschwerdefreiheit. Röntgenologisch findet sich 2 mal ein symptomloses Divertikelrecidiv. MORTENSEN et Al. verfügen über 54 nachkontrollierte Patienten. Nur 7 Patienten klagen noch über Schluckbeschwerden. In 3 Fällen besteht röntgenologisch ein Divertikelrecidiv. 2 Patienten weisen an der Operationsstelle eine Narbenstriktur auf. 2 weitere Patienten klagen über

dysphagische Beschwerden bei normalen Oesophagusverhaltnissen In 1 Fall besteht eine irreversible Heiserkeit infolge einseitiger Stimmbandlahmung MORTENSEN et Al. fordern nicht ausdrucklich die Durchtrennung des m cricopharyngeus, wohl aber die sorgfaltige Freipraparierung des Divertikelhalses, was wahrscheinlich einer Cricopharyngeuszerreissung oder einer Innervationsunterbrechung nahekommt

Von unseren 38 nachkontrollierten Patienten klagen 8 noch uber gewisse Divertikelsymptome und rontgenologisch wird in 7 Fallen ein Divertikelrecidiv gefunden Vorlaufig haben nur 3 dieser Recidivfalle subjektive Beschwerden Allem Anschein nach hangen die Schluckbeschwerden resp die Beschwerdefreiheit vor allem von der Stellung und der Durchgangigkeit des Oesophagusmundes ab Da dem m cricopharyngeus die Oeffnungs und Schliessfunktion des Oesophagusmundes zukommt, ergibt sich daraus die wichtige Bedeutung dieses Muskels in der Entstehung des Divertikelrecidivs und des Divertikels uberhaupt Ist der m cricopharyngeus vollstandig durchtrennt oder seine Innervation endgultig unterbrochen, wie dies wahrscheinlich bei jeder sorgfaltigen Freipraparierung und Darstellung des Divertikelhalses erfolgt, so bildet sich kein neues Divertikel mehr, oder es besteht weitgehende Beschwerdefreiheit Der Grund aller Misserfolge musste damit in einer ungenugenden Durchtrennung resp ungenugenden Freipraparierung und Darstellung des m cricopharyngeus gesucht werden

Die Resultate der zwei bei uns angewandten Operationsmethoden (mit und ohne Durchtrennung des m cricopharyngeus) lassen sich wegen der kleinen Zahlen nur bedingt vergleichen Vor allem im Hinblick auf das oben Gesagte und auf den ESCHER'schen Beitrag erscheint eine Gegenuberstellung trotzdem wertvoll

9 Divertikeltrager sind vor 10—13 Jahren mit Cricopharyngeusdurchtrennung operiert worden Mit zwei Ausnahmen (Falle Nr 32 und 33, wo eine Hiatushernie besteht, die alle Beschwerden erklaren konnte) sind diese Patienten heute beschwerdefrei, obschon in einem Fall (Nr 38) rontgenologisch ein Recidiv festgestellt wird Unter den 26 Patienten, welche ohne Durchtrennung der Cricopharyngeus operiert wurden, sind in 6 Fallen (Nr 24—29) Recidive aufgetreten, wovon allerdings nur 3 Patienten (Nr 26, 28, 29) uber subjektive Beschwerden klagen Die Beschwerden bei Patienten ohne entsprechenden Rontgenbefund sind wahrscheinlich anderen moglicherweise psychogenen Ursprungs

Auf Grund der Erfahrungen, welche in der Berner Klinik an 22 Patienten, und an unserem Krankengut (49 Falle) gewonnen worden sind, ergibt unseres Erachtens die Durchtrennung des m cricopharyngeus sowohl anatomisch als funktionell die besten Resultate, da damit der pathogenetisch wichtigste Faktor eliminiert wird

Zusammenfassung

Aetiologie Klinik und operative Therapie des Zenker'schen Divertikels werden an Hand von Literaturangaben und auf Grund von 38 an der Zurcher Ohren Nasen Hals Klinik operierten und rontgenologisch nachkontrollierten Fallen besprochen Durch die rontgenologische Nachuntersuchung wurden 4 klinisch

symptomfreie Divertikelrecidive entdeckt, während 5 Patienten mit subjektiven Beschwerden röntgenologisch normale Oesophagusverhältnisse aufwiesen. Nachdem ESCHER in seinen Fällen mit durchtrenntem m. cricopharyngeus bei der Röntgennachkontrolle weniger Recidive als wir beobachtet hat, und in unserem Material in der Gruppe mit Durchtrennung des Cricopharyngeus ebenfalls bessere Spätresultate erzielt worden sind, empfiehlt sich die Cricopharyngeusdurchtrennung als Methode der Wahl bei der Resektionsbehandlung des Zenker'schen Divertikels.

LITERATURVERZEICHNIS

ASHERSON, N. Etiology of Diverticulum. J. Intern. Coll. Surg. *20* 531—543, 1953.

AUBIN, A. Ann. d'Oto-Laryng. 1936, 167.

COVEN, M. Surgical treatment of the Zenker Hypopharynx Diverticulum. Pract. Oto-Rhino-Laryng. *21* 129—149, 1959.

DOHLMAN, G. and MATTSON, O. The endoscopic operation for hypopharyngeal diverticula. Arch. of Otolaryng. *71* 744—752, 1960.

ESCHER, F. Zur Therapie der Zenker'schen Divertikel. Schweiz. Med. Wschr. *84* 1073, 1954.

KILLIAN, G. Ueber den Mund der Speiseröhre. Z. Ohrenheilk. *55* 1, 1908.

KIRCHNER, J. A. The motor activity of the cricopharyngeus muscle. Laryngoscope *68* 1119—1159, 1958.

KODICEK, J. and CREAMER, B. A study of pharyngeal pouches. J. Laryng. *75* 406—411, 1961.

LAHEY, F. H. and WARREN, K. W. Esophageal Diverticula. Surg. Gynec. Obstet. *98* 1, 1954.

MORTENSEN, J. D., CLAGETT, O. T., SCHMIDT, H. W. and GRAY, H. K. Onestage Pharyngoesophageal Diverticulectomy. J. Thorac. Surg. *30* 316, 1955.

NEGUS, V. E. Pharyngeal Diverticula. Observation on their Evolution and Treatment. Brit. J. Surg. *38* 129—146, 1950.

SEIFFERT, A. Oesophagoskopie und endoskopische Oesophagus-Behandlung. Arch. Ohren-Nasen-Kehlk. Heilk. *163* 140—171, 1953.

WARREN, K. W. Pharyngoesophageal Diverticulum. Amer. J. Surg. 93 205—217, 1957.

ZENKER, F. A. Diseases of the Esophagus. Cyclopedia of the Practice of Medicine. *8* 46—89, 1878 (New York).

BLEEDING AT TONSILLECTOMY

by

URPO SIIRALA

Helsinki, Finland

As is well known, bleeding at tonsillectomy varies widely from one patient to another The present report, based on statistical study, deals with some possible factors causing this variation The case material consisted of 200 patients subjected to tonsillectomy under local anesthesia

It was studied in an attempt to answer the following questions 1) How is the amount of bleeding at tonsillectomy affected by the suprarenin content of the anesthetic and by the patient's blood pressure, age and sex? 2) What is the relationship between the preoperatively determined bleeding time and the amount of bleeding? 3) Is there any difference between the right and left side from the point of view of bleeding? The results were checked using probability calculus

Material, methods and results

To determine the effect of the suprarenin contained in novocain, in 100 [illegible] sillectomies the right side was anesthesized with 12 ml of 1 % novocain without suprarenin and the left side with the same amount of 1 % novocain suprarenin so- lution This latter was prepared from tablets, each containing 0 1 gm novocain and 0 00025 gm suprarenin Thus each of the sides received 0 12 gm of novocain and the left side in addition 0 00030 gm suprarenin A relatively large [illegible] of anesthetic was injected in the peritonsillar tissue because adequate anesthesia would not have been obtained with a smaller quantity if novocain alone was employed

The patient's general condition, blood pressure and pulse rate were checked before and after the induction of anesthesia Operation consisted in dissecting the tonsils including the capsule — if possible bluntly — from the surrounding tissue, carefully respecting the pillars of the fauces, and finally cutting the tonsils at the base with Brünning's loop

The amount of blood that escaped at operation was measured separately for each side Those cases were excluded in which collection of blood into a measuring glass did not succeed because of the patient's restlessness or for some other reason

Of the operated patients, 64 were males and 36 females They varied in age from 13 to 45 years, the majority being 18 to 28 years old Bleeding from the right side, which was first operated on and anesthesized without suprarenin, totalled 2262 ml, the corresponding figure for the left side being [illegible] ml Thus the average amount of bleeding per patient was 22 62 ml from the right side and

6 91 ml from the left The difference between these figures, 15 71 ml, must be considered to represent the action of suprarenin

Thus the greater amount of hemorrhage on the right side was due in the main to capillary bleeding Four patients had hemorrhage from the wound surfaces after operation The two sides did not differ appreciable as regards postoperative bleeding

To increase the case material, one hundred additional tonsillectomies (59 men and 41 women) were performed, 12 ml of ½ % novocain suprarenin solution prepared from the same sort of tablets than in the earlier series of tonsillectomies was employed bilaterally as anesthetic, the technique of anesthesia, the preoperative preparation, and all observations being the same as above On the basis of the total material of 200 cases, it was possible to calculate the relationship between the amount of bleeding at tonsillectomy on the one hand and the patient's blood pressure, age and sex, and the preoperatively determined bleeding time on the other

The patients were divided into three groups for study of the effect of blood pressure *Group 1* included the patients with a preoperative blood pressure (at most 115 mm Hg), *group 2* those with a preoperative blood pressure (115—130 mm Hg), *group 3* those whose blood pressure exceeded 130 mm Hg It appeared that the average amount of bleeding from both tonsils together was 21 2 ml in group 1, 23 9 ml in group 2, and 32 8 ml in group 3 A statisticomathematical comparison of these figures shows that patients whose blood pressure exceeds 130 mm Hg bleed on an average almost significant more than do those with a low blood pressure, at most 115 mm Hg The average amount of tonsillar bleeding in the former (group 3) seems also to be somewhat greater than in those patients with a normal" blood pressure (group 2)

Determination of the bleeding time — in the case of patients with normal or shorter than normal values — was not found to be of use from the point of view that a patient's greater or slighter tendency to bleeding during operation might be estimated on its basis beforehand

The average amount of bleeding in women was 17 8 ± 2 01 ml and in men 27 5 ± 2 04 ml The difference, 9 7 ml, is statistically significant Bleeding at tonsillectomies of men was thus clearly of larger amount than in the case of women All women were operated on during the intermenstrual period

No distinct tendency for the amount of bleeding to increase or decrease with advancing age was observed

There was no statistically significant difference between the right side and the left It was found that the decrease in bleeding obtained with a ½ % novocain suprarenin solution was almost as great as the corresponding decrease resulting from the use of a 1 % solution containing twice as much suprarenin the difference between average amounts of bleeding was only 2 3 ml

Apart from a postoperative hemorrhage occurring in 4 patients, there was only one major complication bronchopneumonia developed in one of the male patients two days after the operation but it was fairly rapidly cured with medication

Conclusions

1 Peritonsillar injection of novocain solution containing 0 00030 gm of suprarenin (corresponding to about 7 drops of ordinary 0 1 % solution) resulted in a decrease in bleeding at tonsillectomy to about 30 % of what it was without suprarenin

2 Almost the same result was achieved with half of this suprarenin quantity

3 The effect of suprarenin at tonsillectomy seemed to be due chiefly to diminished capillary hemorrhage

4 The suprarenin content of novocain was not found to affect postoperative bleeding to any extent Postoperative bleeding appeared in a total of 4 cases, or in 2 0 %

5 Patients with a low blood pressure bled less than those with a relatively high blood pressure

6 Variations in the length of bleeding time, if it did not exceed the normal value, were not found to be of importance from the point of view of determining in advance the degree of bleeding at tonsillectomy

7 The average amount of bleeding was less for female than for male cases, the difference being statistically significant

8 The different age groups dit not differ appreciably in regard to amount of bleeding

9 There was no statistically significant difference between the right and the left side as far as amount of bleeding is concerned

THE PATHOLOGY OF OTOSCLEROSIS

by

I SIMSON HALL, F R C S E

Edinburgh, England

With the success of Holmgren and Sourdille in evolving a safe and practical method of circumventing the disease of otosclerosis, attention became focussed upon the possibilities of relief of deafness

With a very few exceptions all writings upon otosclerosis were concerned with technical matters, to the virtual exclusion of research into the fundamental cause of the disease

At the same time it is interesting to observe that the high development of the technique of the fenestration operation was responsible for a new phase in observations upon the pathology of otosclerosis, for it provided a lavish supply of material from early active cases of otosclerosis, and rendered observers independent of *post mortem* material which had been hitherto the only and an uncertain source of supply The present writer succeeded in devising a technique of operation by which the ampullary part of the horizontal canal could be removed with all its layers intact for histological examination In this way the whole clinical picture, of history, reactions to test procedures, and future progress of the case came under observation in each individual case

In the early years this work was carried out in close collaboration with Professor Felix Nager in whose laboratory many prolonged discussions were held and to whom the writer owes a great deal for the help and encouragement he received A little later a colleague Dr R F Ogilvie, was taken into the work for it was realised that certain fundamental problems, chiefly connected with the development and evolution of the cell were properly those of the scientific mind fully trained in the fundamentals of pathology Thus a stimulating and fruitful partnership was founded

Early training by Dr J S Fraser, and close association with Professor Nager had engendered an outlook which was eminently traditional that is, that otosclerosis was a disease entity that it occurred like any other disease spread, and finally recovered All writings up to this point by Sibermann, Wittmaack, Manasse Weber and many others took this concept for granted, and the only one to question the idea was Dorothy Wolff working with Julius Lempert

Working with the Edinburgh material, so strong are the habits of thought developed by traditional teaching that it required two years study before it was realised that the appearances seen under the microscope would not and could not be fitted into the standard theory

Some other concept of the origin of otosclerosis had to be found for it was

obvious that the great part of the material available showed evidence of healing and not of spreading disease As this material was taken in the main from young people in the active and early stages of the disease from a clinical point of view, it was impossible to adhere to a theory of a disease spreading in the labyrinth capsule and thereby producing symptoms

Close scrutiny of the literature of the past showed that current theories rested upon slender and mostly insecure foundations, and supposition played a large part in their formation This was undoubtedly due to the fact that post mortem material was the sole source of information Even in Guild's monumental series of temporal bone examinations there was a striking absence of early" changes referable to otosclerosis, and even Professor Nager's superb collection in Zurich could not produce evidence of the earliest stages of otosclerosis It was obvious that Wittmaack must be correct when he stated as his opinion that there must be an early stage of breaking down of bone — a stage of osteoclasis

This appeared in the Edinburgh material in small degree, as it does in the Zurich collection, but it was evident when correlating the clinical signs of increasing deafness with the histological appearances that it was *not* the active spreading disease which caused deafness, but the healing which followed activity

Having therefore discarded the standard theory of the cause of deafness in otosclerosis the search began for some way of discovering what was the earliest recognisable stage of otosclerosis

The close association of otosclerosis and osteogenesis imperfecta in Van der Hoeve's syndrome gave some hope that if a number of cases of new born osteogenesis imperfecta could be examined it was reasonably certain that in at least one case the seeds of otosclerosis would be found The first step in this investigation was to prove that in Van der Hoeve s syndrome it was true otosclerosis which was encountered Owing to the kindness and cooperation of Professor Wullstein this was accomplished, and attention could then be focussed upon early cases of osteogenesis imperfecta

Study of cases of osteogenesis imperfecta, of which we have seven now in our collection, shows that the early changes in this disease and in otosclerosis are indistinguishable There is evident instability of bone which can be seen to be the subject of replacement even before birth, and the process continues in the young adolescent so that it is impossible to distinguish between the two diseases

Studies of the stapedial footplate *in otosclerosis and the* heads of the other ossicles reveal similar changes, showing *that the* disease is not confined to the labyrinth capsule only, but if search *along these lines* were to be made changes might well be found throughout the *skeleton* These latter studies seem to point to the fact that the unique structure *of the* labyrinth capsule has decided the characteristic appearance of otosclerosis *in that region*, while in other bones e g the footplate of the stapes having no *enchondral* layer, the typical blue mantle is not found This may explain why typical *otosclerosis* has never been identified elsewhere in the skeleton

Our researches therefore do not necessarily mean that otosclerosis and osteo-

genesis imperfecta are identical diseases, but that they have at least a common origin in a hereditary insufficiency of the osteoblast which causes early and occasional widespread reconstruction of the bone of the labyrinth capsule

Many associated problems remain to be solved, as for instance why it occurs in the sites in which it is commonly found, but as we have pointed out elsewhere with histological evidence to emphasise it, this is probably the most vulnerable part of the labyrinth capsule to incidental damage as for instance by infection

It has given me the greatest pleasure to write these few words summarising many years of research, to honour the retirement of one of the leading figures in otolaryngological research My association with Professor Eelco Huizinga has been stimulating and profitable, and I wish him many years yet of useful scientific work

RADIOLOGICAL EXAMINATION OF THE LARYNX IN CASE OF LARYNGEAL CARCINOMA AND IMMOBILITY OF THE VOCAL CORD

by

H C STAM *

Groningen, The Netherlands

The treatment of laryngeal carcinoma has always had Huizinga's special interest, patients have come from all over the Netherlands for treatment in his skilfull hands The choice between the various operative procedures or radiotherapy is not an easy one and meticulous investigation of the extent of the tumour is a necessity for the right selection

When Huizinga and I compared the radiograms of patients suffering from laryngeal carcinoma with the clinical data and the specimen from his many operations — which he had kept carefully — we found a very close relationship between analysis of the tumourextent on the radiogram and the actual extent in the specimen, often the radiogram revealed facts unsuspected at indirect or even direct laryngoscopy We thus learned to interpret the data of the radiograms and the right choice in positioning and in laryngeal function during exposure to obtain radiograms yielding optimal information

We found a discrepancy however between some of the specimens and the radiograms taken shortly before operation, the same difference existed between a radio-

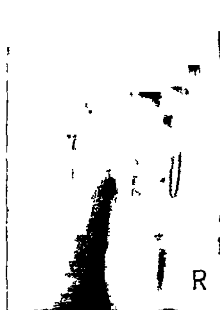


Fig 1 Frontal tomogram of patient with laryngeal carcinoma of the right vocal cord and probable extension in the subglottis



Fig 2 Frontal section trough the specimen without involvement of the subglottic area

* *From the Radiological Institute University of Groningen (Director Prof Dr J R Blickman) Department of Radiotherapy (Head Dr H C Stam)*

gram of the specimen and one of the patient Figure 1 is from a frontal tomogram made during fonation of a patient with this phenomenon. besides enlargment of the right vocal cord extension in the subglottic area probably exists, which is not found in the specimen (figure 2) or in its radiogram however

As subglottic extension of the growth is regarded as a bad prognostic sign and requires special precautions at operation or radiotherapy, the discrepancy found (figure 3) made a closer investigation necessary It became evident that the probable subglottic extension always had the same aspect and was found only in cases in which the involved vocal cord did not move during the change from fonation to breathing and vice versa

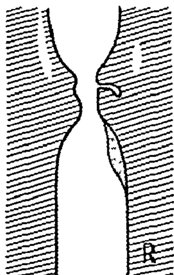


Fig 3 Discrepancy between tomogram and specimen

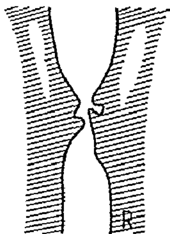


Fig 4 Frontal tomogram during fonation of patient with paralysis of the right nervus recurrens

The radiographic image of the paralysed vocal cord is well known and shows the same aspect of an apparent increase of tissue in the subglottic space, but no evidence of a real swelling can be demonstrated Therefore the image must have a functional basis and probably is due to loss of muscular tone (figure 4) The same radiological symptom, which we have called the "paralytic curve", can be explained in cases of laryngeal neoplasma as an expression of impaired mobility of the vocal cord *and not as an extension of the tumour, the latter, when present* lacks the typical form of the "paralytic curve"

A typical "paralytic curve" was demonstrated in 13 cases, probable existence in 5 cases in which the vocal cord appeared not quite immobile Only two cases with immobile vocal cords did not show a "paralytic curve" different causes for impaired mobility could be held responsible for this phenomenon, but no differentiation could be made between the separate forms of fixation of the vocal cord so far

It is a pleasure to bring the problem of the "paralytic curve" in honour for Eelco Huizinga in the international forum of his many friends

MEDICAL RESPONSIBILITY

by

W H STRUBEN *

Rotterdam, The Netherlands

Extirpation of the larynx is executed far more often today than 30 years ago Although this operation may mean saving the life of a patient, it also means loss of one of the most important forms of human communications

Since the first larynxextirpation performed by Patrick H Watson (Edinburgh 1868), the problem of loss of speech after operation has occupied the thoughts of doctors and patients At the end of the 19th and the beginning of the 20th century most of the surgeons however accepted the loss of voice as a not too important point, for trying to save the lives of their patients was the main point

Sir St Clair Thomson and Colledge (London 1930) report in their classic work on cancer of the larynx how at the end of the last century and in the early 20est the patient without a larynx became a social outcast and was only seen at the many medical meetings as a display of the technical skill of the surgeon Loss of the possibility of speech meant for the patient losing his job and he was therefore no longer possible to provide for his family It was a social and financial catastrophe A patient with carcinoma of the larynx at that period had not much choice He could choose between an early death by cancer or life without voice as a social outcast

The very important change in radiation treatment, as first demonstrated by Coutard in 1922 for the treatment of laryngeal carcinoma, was of little consequence for these patients, as those patients who should be treated by total extirpation were as a rule not fit for curative radiation therapy The number of patients that stayed alive prior to 1920 was little, most of those patients died of infection of the lower airways or from woundinfection

The arrival around Worldwar II of sulfa drugs, antibiotics, general anesthesia, bloodtransfusion and better knowledge of physiology of man brought better chances of survival for our patients Even more important became thus restoration of speech Up till that period now and again a single patient found himself a way of expressing himself in a more or less understandable way Gluck and Gutzmann in 1908 were the first who recognized the importance of restoring the voice of patients who had been treated by extirpation of the larynx

The last 30 years have brought an enormous improvement in speechtraining and follow up on laryngectomees Our knowledge of behaviour of malignant tumours of the larynx our experience and our medical and technical possibilities are much

* *From the Ear Nose and Throat Clinic Dykzigt' Hospital, Rotterdam (Head Prof Dr W H Struben)*

gram of the specimen and one of the patient Figure 1 is from a frontal tomogram made during fonation of a patient with this phenomenon besides enlargment of the right vocal cord extension in the subglottic area probably exists which is not found in the specimen (figure 2) or in its radiogram however

As subglottic extension of the growth is regarded as a bad prognostic sign and requires special precautions at operation or radiotherapy, the discrepancy found (figure 3) made a closer investigation necessary It became evident that the probable subglottic extension always had the same aspect and was found only in cases in which the involved vocal cord did not move during the change from fonation to breathing and vice versa

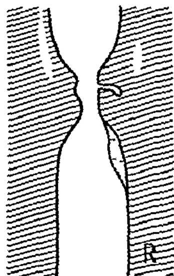


Fig 3 Discrepancy between tomogram and specimen

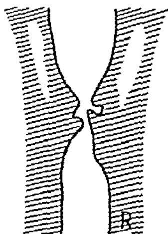


Fig 4 Frontal tomogram during fonation of patient with paralysis of the right nervus recurrens

The radiographic image of the paralysed vocal cord is well known and shows the same aspect of an apparent increase of tissue in the subglottic space but no evidence of a real swelling can be demonstrated Therefore the image must have a functional basis and probably is due to loss of muscular tone (figure 4) The same radiological symptom which we have called the paralytic curve can be explained in cases of laryngeal neoplasma as an expression of impaired mobility of the vocal cord and not as an extension of the tumour the latter when present lacks the typical form of the paralytic curve"

A typical paralytic curve was demonstrated in 13 cases probable existence in 5 cases in which the vocal cord appeared not quite immobile Only two cases with immobile vocal cords did not show a paralytic curve" different causes for impaired mobility could be held responsible for this phenomenon but no differentiation could be made between the separate forms of fixation of the vocal cord so far

It is a plea re t l r ng the problem of the paralytic curve in honour for Eelco Huizin th nternational forum of his many friends

who were not yet pensioned at the time they underwent a laryngectomy, back in their old job In this way there is no more question of being a social outcast, or not being able to provide for their family

It is in Holland Prof Dr Eelco Huizinga and his co-workers Mrs A. J Moolenaar Bijl, Dr P H Damste, Dr Jw van den Berg and others, who in this field have done pioneer work of the greatest importance what has been internationally recognised It has been Huizinga's enormous interest in the well being of his patients and the improvement of their situation, this feeling of responsibility towards his patients, which has started the research on esophageal speech and under his supervision eminent teamwork has resulted in knowledge of the greatest importance

Thanks to Huizinga and his co-workers and others in America, England and Europe the loss of voice is no longer a point of paramount importance by the decision for laryngectomy, because now we have the knowledge that far most of our patients will be able to speak again after the operation and will be able to return to their position in society as a fit person It is nowadays an exception if a patient cannot resume his old job

HEREDITY IN OTOSCLEROSIS

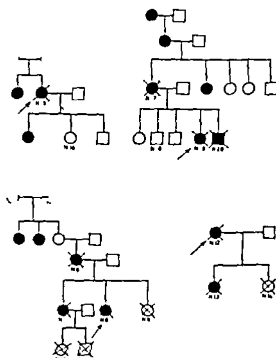
by

JUAN M TATO

Buenos Aires, Argentina

Although the mode of inheritance of otoclerosis has been difficult to establish by statistical and genitical studies of pedigrees, it is known that a genetic factor is involved as the main cause of the disease

The difficulty in the genetical analysis of pedigrees lies in the fact that hearing impairment appears only when the otosclerotic focus reaches the oval window and according to Guild's histological investigations this occurs only in 15 to 35 per cent of the cases



Hoping that cytogenetical studies could help us to detect the histological cases, without clinical manifestations of the disease, preliminary studies has been made in otosclerotic patients and normal relatives Some cells with trisomy and tetrasomy for a chromosome of the group 13—15, probably a number 13, has been found in blood and skin short time cultures, chromosome could be related in some way to the disease but we cannot yet determine if it is a constant alteration in otosclerotic patients

The pedigrees of four families showing a dominant inheritance of the disease and this chromosomal mosaicism may be seen in fig 1 Nevertheless more studies are necessary before any conclusion could be established on this subject

TABLE 1

Case				Diagnosis						Familial cases of clinical otosclerosis	Pedigree No
				Audiogram					Operation		
No	Name	Sex	Age	T	M	P	Unit	Bi			
1	EG	F	28	+	—	—	—	+	+	None	—
2	ND	F	35	+	—	—	—	+	+	„	—
3	HM	M	39	+	—	—	—	+	+	Father and paternal uncle	1
4	CC	M	41	—	+	—	—	+	+	None	—
5	MIZ	F	38	+	—	—	+	—	No	Mother and maternal uncle and aunt	2
6	PLLG	F	66	—	+	—	—	+	No	Two maternal aunts and two daughters	
7	ELLM	F	43	+	—	—	—	+	+	Mother, sister and two sisters of the grandmother	
8	EGLL	F	40	+	—	—	—	+	No	Mother, sister and two sisters of the grandmother	
9	EGLL	F	38	—	—	—	—	—	No	Mother, two sisters, and two sisters of the grandmother	3
10	AEM	M	15	—	—	—	—	—	No	Mother, grandmother, maternal aunt	
11	MCM	F	13	—	—	—	—	—	No	Mother, grandmother, maternal aunt	
12	JRC	F	45	—	+	—	—	+	+	A daughter	
13	MRC	F	23	+	—	—	—	+	No	Mother	
14	MMC	F	21	—	—	—	—	—	No	Mother and a sister	4
15	MBB	F	70	—	—	+	—	—	No	Mother, grandfather, a sister and two children	
16	AB	M	48	—	—	—	—	—	No	Mother, grandmother, maternal aunt, a brother and a sister	
17	MLG	F	41	—	+	—	—	—	+	Mother, grandmother, maternal aunt and a brother	5
18	OB	M	35	+	—	—	—	+	+	Mother and a brother	
19	MER	F	55	—	+	—	—	+	+	A sister and a daughter	
20	MJR	F	27	—	—	—	—	—	No	Mother and maternal aunt	6

THE ETIOLOGY OF CHRONIC SUPPURATIVE OTITIS MEDIA

by

T G WILSON

Dublin Ireland

It is a great privilige to be invited to contribute to this special number Professor Eelco Huizinga is undoubtedly one of the greatest otolaryngologists of our time This is not the place to enumerate the many scientific achievements stemming from his great gifts of intellect, but it may be permissible to thank him for the way in which he has helped our specialty and its individual members His is indeed a strong, kindly and generous personality

In a comparatively short period Professor Huizinga has seen many changes in the practice of our specialty, for a few recent decades have seen more alterations in the character of disease than the whole of the preceding century The tide of bacterial infection has receded and viral neoplastic and degenerative diseases have assumed increased importance This is largely due to the use of antibiotic and chemotherapeutic drugs and it is probably also partly accounted for by a cyclical diminution in the virulence of the organisms themselves Improved standards of living must also play a big part in the virtual disappearance of many of the acute pyogenic infections which were so troublesome a generation ago

Suppurative otitis media acute and chronic, is a case in point While chronic suppurative otitis is still dangerous in its deceptively mild and insidious way, acute otitis media is now seldom seen in adults and is more than ever a disease of children who are of course particularly liable to otitis media for reasons mainly anatomical and immunological Under the former heading are the soft bones open sutures and short patent eustachian tube of infancy, and the remains of mesenchymal tissue in the middle-ear cleft

The severity of the disease in children appears to vary directly with the social status of the child Of 346 children seen in a private general otolaryngological practice in Dublin during the years 1959 61 62 showed signs of minor tympanic infection but did not perforate 8 perforated or required myringotomy, but subsequently healed rapidly and 3 required major mastoid surgery

These figures may be contrasted with those obtained in a hospital practice in which most of the patients come from a considerably lower income group Here the figures for acute otitis are not dissimilar but in chronic otitis media a very different picture is presented Of 683 children attending the otolaryngological clinic of the National Children s Hospital Dublin in 1961, 51 presented with discharging middle ears Of these children 22 required mastoidectomy in some form One had a cerebellar abscess one had facial paresis on admission and one developed pneumococcal meningitis An interesting case which did not come to operation was a child of twelve from whom a pedunculated polyp of the postero-

superior drum margin had been removed five years previously Section then showed typical cholesterol granulomatosis After the polypus had been removed the child developed a postero superior perforation with profuse otorrhoea She was then operated upon for a ventricular septal defect, and received only conservative treatment for five years At the end of this period the ear was found to be quite dry, the tympanic membrane was soundly healed but had considerable trabeculated scarring in the postero-superior quadrant, and the hearing was very nearly perfect

These two series are too small to allow of valid statistical conclusions but the contrast between the public" and private" patients is striking, because of the considerably greater number of chronic and complicated cases amongst the former The more affluent group develop otitis media fairly frequently, but when they do the condition is often of a minor nature and clears up rapidly without leaving after-effects Obviously this must be due to their receiving better general medical care and hygiene

This immediately raises the question of the validity of the views held by many authorities, from Wittmaack to Ruedi that chronicity may often result from a silent neonatal otitis media due to the aspiration of foreign matter such as vernix caseosa and meconium while still in utero or from the infection of mesenchymal remnants in the middle ear These theories do not explain why the children of well to-do parents are so much less likely to develop chronic otitis media than their poorer cousins Nature nurtures the embryos of the rich and the poor alike, sometimes at the expense of the poor child s mother It must, however, be admitted that it is difficult to explain otherwise a group of cases fairly frequently seen in hospital practice, of babies who suffer three or four attacks of acute otitis media with perforation of the membrana tensa during the first year of life

Why does otitis media become chronic? The question has been discussed many times Conditions such as the presence of a persistent perforation in the tympanic membrane and latent mastoid infection have been cited but surely these amount to a confirmation of the state of chronicity rather than an explanation of how that state came to exist

The following classification is suggested

1) Infection following congenital causes such as intra uterine irritation of the middle ear tract and the presence of mesenchymal remnants in the middle ear

2) An acute necrotizing infection (Ruedi 1958) of the middle ear in infancy

3) A slow insidious infection of the middle ear

4) A combination of (1) with (2) or (3)

In my opinion (3) is much the commonest and is greatly abetted by lack of hygiene and unsuitable living conditions

Chronicity may be established very early often during the first months of life It is important therefore that the disease should be attacked early and actively

THE ETIOLOGY OF CHRONIC SUPPURATIVE OTITIS MEDIA

by

T G WILSON

Dublin, Ireland

It is a great privilige to be invited to contribute to this special number Professor Eelco Huizinga is undoubtedly one of the greatest otolaryngologists of our time This is not the place to enumerate the many scientific achievements stemming from his great gifts of intellect, but it may be permissible to thank him for the way in which he has helped our specialty and its individual members His is indeed a strong, kindly and generous personality

In a comparatively short period Professor Huizinga has seen many changes in the practice of our specialty, for a few recent decades have seen more alterations in the character of disease than the whole of the preceding century The tide *of bacterial infection has receded and viral, neoplastic and degenerative diseases* have assumed increased importance This is largely due to the use of antibiotic and chemotherapeutic drugs, and it is probably also partly accounted for by a cyclical diminution in the virulence of the organisms themselves Improved standards of living must also play a big part in the virtual disappearance of many of the acute pyogenic infections which were so troublesome a generation ago

Suppurative otitis media, acute and chronic, is a case in point While chronic suppurative otitis is still dangerous in its deceptively mild and insidious way, acute otitis media is now seldom seen in adults and is more than ever a disease of children who are, of course, particularly liable to otitis media for reasons mainly anatomical and immunological Under the former heading are the soft bones, open sutures and short patent eustachian tube of infancy, and the remains of mesenchymal tissue in the middle-ear cleft

The severity of the disease in children appears to vary directly with the social status of the child Of 346 children seen in a private general otolaryngological practice in Dublin during the years 1959 61 62 showed signs of minor tympanic infection but did not perforate 8 perforated or required myringotomy, but subsequently *healed* rapidly and 3 required major mastoid surgery

These figures may be contrasted with those obtained in a hospital practice in which most of the patients come from a considerably lower income group Here the figures for acute otitis are not dissimilar but in chronic otitis media a very *different picture is presented Of 683 children attending the otolaryngological* clinic of the National Children s Hospital, Dublin in 1961, 51 presented with discharging middle ears Of these children 22 required mastoidectomy in some form One had a cerebellar abscess one had facial paresis on admission and one developed pneumococcal meningitis An interesting case which did not come to operation was a child of twelve from whom a pedunculated polyp of the postero-

super or drum margin had been removed five years previously Section then showed typical cholesterol granulomatosis After the polypus had been removed the child developed a postero superior perforation with profuse otorrhoea She was then operated upon for a ventricular septal defect and received only conservative treatment for five years At the end of this period the ear was found to be quite dry the tympanic membrane was soundly healed but had considerable trabeculated scarring in the postero superior quadrant and the hearing was very nearly perfect

These two series are too small to allow of valid statistical conclusions but the contrast between the public and private patients is striking because of the considerably greater number of chronic and complicated cases amongst the former The more affluent group develop otitis media fairly frequently but when they do the condition is often of a minor nature and clears up rapidly without leaving after-effects Obviously this must be due to their receiving better general medical care and hygiene

This immediately raises the question of the validity of the views held by many authorities from Wittmaack to Ruedi that chronicity may often result from a silent neonatal otitis media due to the aspiration of foreign matter such as vernix caseosa and meconium while still in utero or from the infection of mesenchymal remnants in the middle ear These theories do not explain why the children of well to-do parents are so much less likely to develop chronic otitis media than their poorer cousins Nature nurtures the embryos of the rich and the poor alike sometimes at the expense of the poor child s mother It must however be admitted that it is difficult to explain otherwise a group of cases fairly frequently seen in hospital practice of babies who suffer three or four attacks of acute otitis media with perforation of the membrana tensa during the first year of life

Why does otitis media become chronic? The question has been discussed many times Conditions such as the presence of a persistent perforation in the tympanic membrane and latent mastoid infection have been cited but surely these amount to a confirmation of the state of chronicity rather than an explanation of how that state came to exist

The following classification is suggested

1) Infection following congenital causes such as intra uterine irritation of the middle ear tract and the presence of mesenchymal remnants in the middle ear

2) An acute necrotizing infection (Ruedi 1958) of the middle ear in infancy

3) A slow insidious infection of the middle ear

4) A combination of (1) with (2) or (3)

In my opinion (3) is much the commonest and is greatly abetted by lack of hygiene and unsuitable living conditions

Chronicity may be established very early often during the first months of life It is important therefore that the disease should be attacked early and actively

The predominent organism at present is the *staphylococcus aureus*, which was found in 102 of 171 cases examined in the National Children's Hospital in 1960 61 (Jaswon, 1962) Of the *staphylococcus aureus* strains 89 percent were sensitive to erythromycin, which would seem to be the antibiotic of choice Infants who have suffered two or three or more attacks of purulent otitis media with recurrent perforations of the membrana tensa should have the mastoid opened through a post aural incision Any fluid which may be found in the mastoid antrum should be sucked out without disturbing the tympanic contents and the wound closed without drainage Cases which have already become chronic will require more radical surgery Attention to the nose and throat may be necessary in chronic cases but in our experience will seldom result in a dry ear

The principal conclusions from this necessarily short survey are therefore that chronicity in otitis media is arrived at very early, often during the first months or year of life, and that general hygiene and nutritional conditions may play a considerably greater part than congenital predisposition in establishing the state of chronicity It is suggested that during the first year of life a simple cortical mastoidectomy may often be of value in arresting the disease

REFERENCES

JASWON N N (1962) Personal Communication
RUEDI L (1958) J Laryng 72 593

★

COVER
PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

From the Department of Otolaryngology Harvard Medical School and the Massachusetts Eye and Ear Infirmary Boston

ACTA OTO LARYNGOLOGICA

S U P P L E M E N T [illegible]

RADIATION AND EAR

BY

GEORGE KELEMEN

ACTA OTO LARYNGOLOGICA KARLAVÄGEN 41, STOCKHOLM O

From the Department of Otolaryngology, Harvard Medical School and the Massachusetts Eye and Ear Infirmary, Boston.

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 184

RADIATION AND EAR

Experimental Studies

BY

GEORGE KELEMEN

UPPSALA 1963

Aided by grant AT (30-1)-2712 of
the U. S. Atomic Energy Commission and
grant no. 301-1[illegible] of the Central Bureau of Research of
the American Otological Society, Inc.

PRINTED IN SWEDEN BY
Almqvist & Wiksells
BOKTRYCKERI AKTIEBOLAG
UPPSALA 1963

CONTENTS

Desjardin (1931) in a critical survey on the action of x ray and radium on the ear remarked that the influence of x rays had received scant experimental attention—possibly because no evidence of injury to the auditory apparatus of humans has ever been reported—and that investigation of the action of radium had been confined to the labyrinth. Girden & Culler (1934) remarked that while investigation upon the biological effect of roentgen rays ranged from studies on the single cell to studies on a complete human system very little became known with regard to their effects upon sensory processes. As late as 1960 Clemenson & Nelson felt induced to write that the only sense organ of any interest as far as radiation is concerned is the eye.

All this is the more remarkable as, clinically, there is no part of the ear which has not been investigated and treated by x ray or radium claiming in the superabundant literature sometimes far reaching effects. Generally, in reports enumerating changes in various parts of the organism no attention has been paid to the organ of hearing. The comparatively few publications that have dealt with ear and radiation as the main or the only topic are registered in the following survey without claim to giving an exhaustive list.

X-RAY AND EAR

(a) *Experimental investigations*

Thielemann (1928) is first to be mentioned here. After applying x rays to white mice capillary hyperemia in the middle and the inner ear was seen increasing in intensity with higher doses until perilymphatic hemorrhages appeared chiefly in the basal turn of the cochlea and the region of the cristae, rarely in the maculae. Serous exudation, seropurulent secretions and scattered hemorrhages represented the reaction in the middle ear.

Girden & Culler (1933, 1934, 1935) examined the auditory effects of x rays in dogs conditioned to respond to a given tone and exposed to a 1000 cycle sound. Radiation was continued for about 4 months until the total cumulated dosage for one of the dogs reached 11 000 and for the other 4 500 r. A transient increase in auditory acuity followed and lasted for some 4 weeks. Eight dogs received smaller doses. Betterment of hearing followed, usually after a latent period of 11 days. Relatively larger doses resulted in shorter periods of increased hearing acuity.

Hertwig (1941) studied the organ of hearing in x-ray mutation of circling mice. Several parts of the ear were malformed but no one of them could be pointed out definitely to be the source of the abnormal movements.

Novotny (1951) treated six guinea pigs in a single experiment with a total dose of between 1600 and 7200 r. 6 weeks after radiation no animal showed adverse effects except for reduction of the cochlear potentials in all six with a maximum around 4000 Hz corresponding to ca. 8.4 db. The non-radiated side, vitally fixed, served as control. Microscopically up to 5600 r both sides remained free of lesions and any reported by previous writers were considered due to some artificial factor.

Hicks (1953) produced malformation in rats through radiation. Exposure on the 12th fetal day resulted in irregularity of the inner ear in its bony configuration with slight misplacement ventralward.

Gerstner, Konecci & Taylor (1954) selected 20 guinea pigs with a strong pinna reflex and exposed them to 8000 r. Between cps of 860 and 6400 rise of the reflex threshold became evident after 4 hours, within 24 hours the pinna reflex was completely abolished.

Kelemen (1955) investigated histologically the litter of albino rats exposed in experiments by Hicks to x-rays. The sensory end organs of the inner ear were found structurally intact and in appropriate developmental stage but amid extensive hemorrhages in their vicinity and against a background of severe radiation-induced endocranial deformities.

Ivanov (1957, 1958) exposed rabbits and guinea pigs to local (2000 r) and total body (600 r for rabbits, 300 r for guinea pigs) radiation. Under the microscope sharply pronounced atrophic and degenerative changes showed up in the inner ear with hemorrhages in the middle and inner ear. A number of rabbits developed labyrinthitis with corresponding clinical symptoms and histological changes.

Pautov (1958, 1959) studied wound healing in guinea pigs and rabbits subjected to x-rays. The tympanic bulla was fenestrated, the auricle was amputated, different plastic procedures were executed. In the guinea pigs at dissection the fenestration defect was closed by scar or bone tissue in the radiated as well as in the non-radiated ear. In the rabbits amputation was not followed by complications; at plastic interventions bleeding was inconspicuous but tissue elasticity was diminished.

Kozlov (1959) lost many of his 154 guinea pigs by radiation sickness—about 50 per cent at a dose of 350 r. Bio-electric currents were investigated by sounds between 500 and 8000 Hz, loudness 120 db, tension 4 volts. At the height of radiation sickness the hearing decreased, as shown by lowering of the auricular reflex and the bioelectrical potential of the cochlea to all frequencies between 500 and 8000. Under the microscope in the decapitated or *in vivo* fixed animals small hemorrhages were noted in the middle and the inner ear. No changes were observed in the sensory or nervous apparatus of the inner ear. The morphological changes were interpreted as vascular damage with changes in the biochemical characteristics of the cellular fluids.

Miani & Marucci (1959) found, in the horse ciliary activity in the Eustachian tube unchanged after submitting excised parts of the mucous membrane to x rays

Murphy & Harris (1961) in the rat, found the hearing, after x ray radiation to the head, unchanged

Guggenheim and collaborators (1961) investigated the effects of x rays on the tympanal mesenchyme cycle in the rat A single dose was administered to the right ear, the left serving as control In the active phase (birth through 11th day) no significant effect as to retarding the growth was produced nor, in the subsequent period of decline, was involution hastened

Berg & Lindgren (1961) exposed, in rabbits, the right half of the skull to x rays, with the left half shielded At histological examination they found that "An analysis of the evaluation of damage to the internal ear and middle ear was made difficult by the high frequency of complications due to infection in some groups But the role of this all important fact was disregarded in the listing of changes in the middle and the inner ear, not different from those which could have been found in control animals or indeed in the left, non radiated ears

(b) *Observations in humans*

Berberich (1935) found an asterisk shaped perforation of the tympanic membrane after x ray treatment for carcinoma, and observed symptoms of irritation of the vestibular apparatus, clearing after a few days

Work (1950) saw aseptic necrosis with overlying ulcerations of a portion of the bony external canal produced by x ray

Block (1952) described in operative material from the temporal bone following x ray therapy, a non specific osteomyelitis with intensive metaplasia of the bone

Moskovskaya (1959) examined patients after x ray treatment for malignancies Prolonged deep changes in the central nervous system were reflected in affections of the vestibular analyzer (term introduced by Pavlov) Increased rotatory and caloric irritability was interpreted not so much as a direct ionizing effect on the peripheric organ as a consequence of decrease in cortical inhibition

Gyorkey (1960) saw, after massive x ray radiation to the head and at exploration because of hearing loss the long process and the lenticular process of the incus replaced by a fibrous band

RADIUM AND EAR

(a) *Experimental investigations*

Ewald (1905) with radium, had preceded the x ray experimentation (Tuckmann 1928) by 23 years Glass beads containing about 3 mg radium

bromide were placed, in pigeons, through a small opening in the skull in the vicinity of the labyrinth for a few hours. Empty glass beads were applied in controls. Turning of the head and other manifestations as seen after removal of the labyrinth followed. The disturbances developed several days after exposure, proportional in intensity to the duration of the exposure.

Marx (1909) applied glass capillaries filled with 4 mg radium bromide to his animals: in pigeons, directly over the foramen communicans through an opening into the ear capsule, in guinea pigs directly on the cochlea through a wide opening in the middle ear. The pigeons remained completely normal; the guinea pigs showed no changes at examination of the auricular reflex with the continuous line of frequencies. The pigeons were decapitated within 2½ months after exposure; microscopically, simple atrophy of the sensory organs was noted. The guinea pigs were decapitated between 2½ and 6 months after exposure. More or less pronounced signs of degeneration and atrophy were seen. The microscopical lesions were given in great detail, with differences in the findings attributed to variations in the positioning of the radium.

Chilov (1927) wanted to separate the action of the maculae and cristae, assuming that radium will eliminate the activity of the otoliths and leave the cristae intact. Radium emanation was placed into the tympanic cavity of cats. Severe vestibular disturbances followed. Microscopically, labyrinth damage was seen in different degrees of severity. The conclusion was that radium disturbed the function of the semicircular canals, the utricle and the cochlea but did not influence the saccule.

Nylen, Engfeldt & Larsson (1959) applied to 20 rats a 2 mm wide 185 MeV proton beam with the purpose of elaboration of a locally applicable microsurgery. Two weeks later changes manifested themselves in the form of a peristapedial edema; eight weeks later, degenerative changes followed in the acoustic nerve just inside the internal acoustic orifice; no pathology appeared in the inner ear or other parts of the temporal bone. Twelve weeks later circumscribed damage to bone and muscle outside the middle ear appeared, with part of the bone completely necrotized.

Mzani (1959) found that, in the horse, radium was unable to disturb the ciliary activity in the tubal mucous membrane.

Mzani & Marcucci (1960) applied beta and gamma rays of 198 Au to this mucous membrane without effect.

Zlotnikov (1961) placed radium emanation into the bulla of rabbits in doses between 13 and 25 mc, except in one case where 34 mc were used. A typical vestibular reaction followed: inhibition of the postrotatory and position nystagmus, signs of a lesion in the vestibular centers, particularly in the nucleus of the vestibular nerve. Histology showed hemorrhages in the labyrinth with degenerative changes in the organ of Corti, in the cristae and the maculae, extending into the vestibular nucleus.

(b) *Observations in humans*

Berberich (1936) found with radium rays penetrating to the labyrinth sequelae similar to those experienced after x rays (cf above)

Work (1950) described lesions of the external auditory canal produced by radium similar to those following x ray radiation

Borsanyi Blanchard & Thorne (1961) observed the ears of patients under going radiation with Co 60 for head and neck tumors A small shift in the hearing threshold was attributed to development of a radiation otitis media perceptive hearing loss with temporary recruitment was thought to be related to a transient vasculitis in the inner ear

MATERIAL AND METHODS

The observations were carried out on two groups of rats

The first comprised 90 young rats of the Sprague Dawley strain with a body weight between 100 and 120 g In the investigations of Dr Julian L Ambrus Roswell Park Memorial Institute Buffalo N Y whose collaboration in releasing this material is greatly appreciated X rays were administered at 2 megavolts 1 milliampere target distance in air 90 cm Grouping dose and date of decapitation are shown in Table 1 Warren (1958) explained that the response of cells to injury by ionizing radiation does not vary qualitatively with the type of radiation

The second group was made up of 55 rats Charles River Albino strain SD (Sprague Dawley descendent) and CD (Cesarean derived) They were obtained when 21 days old immediately after weaning The reason for acquiring them that young was to avoid as far as possible the non experimental infections that show up in animal farms in increasing frequency with advancing age In this group cobalt was chosen as radiation source

TABLE 1 *Roswell Park group Postradiation days of sacrifice, groups of five each*

In tables 1–6 the controls were omitted

	Days				
Dosage	1	2	3	4	5
100	1	3	8	14	
400	1	3	8	14	18
800	1	3	8	14	
1500	1	3			
3000	1	3			

Dr Laszlo Pinter from the Department of Medical Physics Massachusetts General Hospital extended his most welcome collaboration using a 60 gamma 1 33 MeV bomb The only source of change in dose rate was a different duration of the exposure The question whether the rays should be directed to the ear with shielding of the remaining regions or whether total body radiation should be chosen was decided in favor of the latter procedure cobalt gamma rays passing through with no change of the dose before or after the body is penetrated The animals were enclosed in a brass capsule with walls 1 cm thick Within the capsule they were not further restrained they could even turn around completely in the cylindrical container

From the first group five and from the second group 10 rats were left untreated to serve as controls

DOSAGE

The total body lethal dose in rats without age difference is sometimes assumed to be around 7000 r but with practically no limit when only selected parts of the body are attacked In the present investigations total body radiation was applied with dosage of 300 500 800 900 1000 1200 and 2000 For the fact that dosage level did not make much difference in the date of development of radiation sickness—and so of survival time—one possible explanation is that the animals underwent the radiation in a different state of health

While the animals of the first group were sacrificed by decapitation in the second group decapitation or perfusion with Heidenhain Susa solution was used Perfusion gives doubtless better histological pictures on the other hand after a thorough perfusion the solution does not remain confined to the vascular bed and pathological secretions may be flushed out the result being a deceptive microscopical picture The rats of the first group were shipped in formalin and the same solution was used for fixation after decapitation in the second group When spontaneous death occurred—e g at night—some hours may have passed between death and fixation

After embedding in celloidin the plane of serial sectioning at 15 to 25 μ was a horizontal one i e parallel to the main plane of the cranial base A minor part was sectioned in the frontal plane Right and left ear were processed in a single block Stains used were hematoxylin eosin and Heidenhain Mallory with occasional auxiliary stains like Gomori impregnation such as mounted but left unstained were examined in polarized light or under phase illumination Generally every tenth section was prepared in this manner

The histological work was done by Miss Dorothy Linden and Miss Helen Stewart the photography by Mr Richard W St Clair

NON-EXPERIMENTAL PATHOLOGY

It is obvious that great difficulty will arise by spontaneous or non experimental pathology against which all histological findings must be plotted Previous experience (among others Kelemen & Sargent, 1946 Kelemen, 1951, 1958 Friedmann, 1955, Berberich & Kelemen, 1958) provided ample material to foresee obstacles along this line In these publications manifestation of pathology in the upper airways and the ear was followed in all detail, in this way pitfalls from this angle were fully anticipated The reason e g to choose as material the fetus in work on the problem of ear and radiation was to exclude non experimental pathology, as a factor of paramount importance (Kelemen 1955)

Examination of five rats of the Roswell Park group by serial sectioning *of the nasal and paranasal cavities (radiated with 4000 r)* added the following to former experiences

Main cavity and maxillary sinuses free	2
Main cavity free, maxillary sinuses slight secretion	2
One side of main cavity plus maxillary sinus completely filled by pus, other side main cavity filled, maxillary sinus free	1

This sample shows that occurrence of infection in the upper airways is *utterly unpredictable as to location and distribution of pathological products* or, indeed, as to their occurrence at all

Omitting the controls the proportion of the animals found free of infection is given in Table 2 Altogether, with 130 rats and among 260 ears 84 or 32 3% *were found free of infection*

TABLE 2 *Animals free of infection*

	Free of infection	
	Both ears	One ear
Roswell Park	13 (15 3%)	13 (15.3%)
Charles River	15 (33 3%)	15 (33 3%)

The following Tables 3 4 and 5 show that infection was not influenced either by the dosage of radiation or by *the total number of days, before and* after radiation spent in the farm

Among the 10 controls of the Charles River group with 17 to 22 days in the farm three were free of infection in both ears, and one on one side

The expectation of finding *more healthy animals among* the very young ones was justified as their percentage, in the Charles River group, was double that found in the Roswell Park group

The difficult task of definitive tabulation of the extant changes *into pre-*

TABLE 3 *Roswell Park group Number of animals free of infection after radiation, according dosage*

Dosage	Both ears free	One ear free
100	3	3
400	3	6
800	3	1
1500	4	1
3000		2

TABLE 4 *Roswell Park group Number of animals free of infection after radiation, according post radiation time*

Days after radiation	Both ears free	One ear free
1	3	1
3	7	10
8	1	
14	1	2
18	1	

TABLE 5 *Charles River group Number of animals free of infection after total number (pre and post radiation) of days at the farm*

Total days in farm	Both ears free	One ear free
6	2	1
10	5	1
17	1	2
18	2	4
21		1
30		1
31	2	
34	1	
45		1

viously present and radiation induced can be approached in the following way

Changes in the hearing organ can be reliably classified into chronic and acute the recent can again be split from the acute group with reasonable security By making this categorization more detailed, a more reliable instrument can be created The pathological picture was divided into nine degrees which were then affixed to each animal in the individual protocols

1 *Both ears free of infection*
2 One ear free, other serous infection
3 One ear free, other suppuration
4 One ear free, other suppuration with organization
5 Both ears serous infection
6 Both ears suppuration
7 Both ears suppuration with organization
8 One ear serous labyrinthitis
9 Both ears serous labyrinthitis

Among the publications enumerated in the introductory review only a single one (Berg & Lindgren) mentioned the factor of non experimental infection In the present investigations, in full awareness of its impact, this factor was considered thoroughly in all interpretations

This obstacle may *probably be eliminated by introduction of germfree bred* animal material, at least for experiments of short post germfree duration Similar material proved to be completely free of infection (Kelemen, 1960, 1962) Availability seems to be only a question of time and will greatly facilitate arrival at reliable conclusions

Considering the number of variants entering the picture, a clear tabulation of the observations is almost impossible The controls themselves have to be divided into infection free and infected Artificial damage during the *processing must* be heeded There are differences to observe according to the occurrence of natural death, decapitation or perfusion, the latter after a planned lapse of time or under the pressure of imminent radiation death *time spent at the farm before radiation, exposing to new infections,* time between radiation and death, spontaneous or by sacrifice, finally—and the only factor without interference from the outside—dosage Other variables included unexpected death in anesthesia, which changed a planned perfusion to decapitation it should be added that right and left ears may show entirely

TABLE 6 *Charles River group Survival according dosage*

Groups of five animals each

Dosage	Lived days after radiation	Days in farm before radiation
300	3	18
500	6	11
500	20–30	15
600	4–17	20
900	3	7
900	3	3
1000	4	11
1200	3	31
2000	4	6

different conditions this circumstance pointed to preradiation genesis as radiation would hit both ears simultaneously with identical effect

Survival according to dosage and time are shown in Table 1 (Roswell Park) and Table 6 (Charles River group)

In the Roswell Park group (Table 1) some batches of five lost some of their number by radiation death The Charles River group showed a less uniform picture as more animals succumbed to radiation death probably being less resistant at their tender age They were sacrificed when signs of approaching end became evident most conspicuous being mottling of the fur

Only after taking due notice of all these determinants can one proceed to appraise any change as resulting from the experiment

OBSERVATIONS

Keeping the enumerated variants in mind the single anatomical elements of the ear can present very different aspects within the same ear

The main tabulations were based on 84 infection free ears with the remaining ones furnishing additional pertinent data

CONTROLS

Five rats in the Roswell and 10 in the Charles River group represented the controls Among the 10 ears of the first three and among the 20 ears of the second group 10 were free of infection The higher proportion in the second group may have been correlated to the younger age In these 13 animals the ampullar cristae were normal in all cases and so were the maculae The organ of Corti was clear in one case only in the apical turn in another case it was poorly preserved in all turns Elsewhere it was found *in satisfactory condition albeit in less perfect preservation than the vestibular* end organs

In view of the observations reported later it was of consequence that all 13 were free of hemorrhages the first group after decapitation the second after perfusion Once in a perfused animal a small amount of blood was seen in the niche of the round window In the controls with infected ears the mass of more recent secretions filling the middle ear was sometimes slightly mixed with blood which however was not seen in the tissues (cf with Kelemen & Sargent infrequent non experimental presence of extravasates in the nasal and paranasal cavities of rats)

Where desirable it was possible to draw on a considerable number of animals from other projects to obtain a wider basis for comparison with normal relations as well as with pathological products

ANIMALS FREE OF INFECTION

As most characteristic, instructive pictures could be expected in animals exposed to the highest dosages these will be reported first in this way changes in the other groups can be related to them

In the Roswell group nine rats received 3000 r surviving this dose one day or two days Stretching the criterion infection free to include ears with minimal scattered secretions nine ears belonged to this group The cristae were in good or slightly disorganized condition and so were the maculae —the latter showing sometimes detachment of the otoconia a condition encountered in normals not too infrequently Perilymphatic edema was noted repeatedly with considerable compression of the endolymphatic spaces sometimes depressing the cristae Crista and its cupula showed generally identical conditions giving no reason to declare the latter as more vulnerable The organ of Corti was in most cases disorganized if not completely destroyed with scattering of the other elements of the cochlear duct Most conspicuous was the comparative tenacity of the papilla in the apical turn Here preservation with clearly distinguishable hair was several times seen next to disorganization in the middle and complete disappearance in the basal turn The general picture of destruction was not a flat papilla or empty basilar membrane seen e g after acoustic trauma, but a blown up " condition like acute edema with scattering of the elements The stria suffered the same structural disorganization while the tectorial membrane was thrown against the membrane of Reissner The latter was always intact and without exception in standard position The basilar membrane was clearly drawn and undamaged

The Charles River group offered five rats after 2000 r with an average weight of 47 3 g They were sacrificed in ultimis at the fourth day having spent altogether 10 days at the farm Among the 10 ears seven were free of infection Cristae and maculae were either practically intact or merely loosened up by edema crista and its cupula were in identical conditions. The organ of Corti was everywhere in advanced dissolution, so were the elements of the stria The displaced tectorial membrane was clearly outlined, the basilar membrane intact Reissner s membrane was in standard position A small amount of blood was present once in the middle, once in the inner ear The papilla of Corti was blown apart its constituents scattered within the scala tertia

Ten rats of the Roswell group received 1500 r and died or were decapitated one or two days after administration Among the 20 ears seven were free of infection and yielded the following Cristae and maculae were in good condition with some of the cupulae compressed by perilymphatic edema The latter was the most conspicuous finding in the vestibular labyrinth, at some points in a degree to compress the endolymphatic space to a narrow slit The depressed cupulae were bent but intact The papilla of Corti was in

almost all cases depressed in the basal turn and showed less and less damage ascending until in the apical turn the picture became normal. In one case the contents of the cochlear duct were destroyed with the elements scattered around as if by acute edema. Reissner's membrane was, with negligible deviations both ways, in normal position. Except for a single case where it was destroyed with the rest of the contents of the cochlear duct, the tectorial membrane was intact; so, in all cases, was the basilar membrane.

Four rats of the Charles River group with an average weight of 162 g and having spent 34 days at the farm died or were decapitated at the 3rd day after having received 1200 r. Among the 10 ears eight were free of infection. The cristae were loosened up, at one point compressed by perilymphatic edema; the maculae showed somewhat more resistance; the organ of Corti was destroyed once by hemorrhage, otherwise as by edema scattering its elements. Reissner's membrane was everywhere in normal position, intact, as was the basilar membrane. Contours of the organ of Corti were sometimes still outlined, but invariably in the apical turn. Among the four animals three carried blood within the cochlea.

When it became obvious that the rats were unable to withstand these high dosages they were decreased to a degree where an effect could still be expected. In the Roswell Park group this meant 100, 400, 800, and in the Charles River group 300, 500, 800 and 1000 r respectively.

The Roswell group offered 20 rats exposed to 100 r. Five were free of infection, one side or both sides. Among these one was omitted for technical reasons (paraffin having been found unsuitable). The four rats with uninfected ears were sacrificed on the 2nd post-radiation day. The ampullar cristae were everywhere normal and so were the maculae—the latter, however, frequently with detached otoconia. Many sections presented a normal organ of Corti in the apical turn while in the lower turns much dissolution was seen. A flattened mound was nowhere present; the destruction was explosion-like, as by edema, with the tectorial membrane blown against the membrane of Reissner. The latter was without exception in normal position while the basilar membrane participated, in spots, in the general destruction. One case showed on one side a small amount of blood in the tympanic scala.

The next step was radiation with 300 r. The Charles River group offered five rats (average weight 99.5 g). They died in anesthesia three days after radiation after spending between 21 and 30 days at the farm. Three ears were free of infection—with a fourth added here in spite of a small amount of transudate and blood in the middle ear. All sensory end organs appeared normal.

Twenty-five rats of the Roswell group received 400 r and among the 50 ears 12 were free of infection. Seven animals were killed in the first 2 days. After rejecting one for technical reasons, six showed normal cristae and maculae, the latter sometimes with detached otoconia. The organ of Corti was normal in three and destroyed in the remaining cases; however, in two cases it was [illegible]t in the apical turn. At destruction it was common to find the

tectorial pushed up to the Reissner membrane with the latter in every case in normal position After 14 days one animal showed normal cristae and maculae in both ears normal organ of Corti in one ear and a destroyed organ in the other ear with the tectorial contacting the Reissner membrane, which was in the standard position In one ear of an animal which lived for 18 post radiation days cristae and maculae were normal while the cochlea was lost in the preparatory process

Ten rats of the Charles River group received 500 r and among the 20 ears five were free of infection This low rate could be explained by their comparatively long stay at the farm although this is contradicted by other instances (e g the 1200 r group) One rat (weight 102 g perfused 6 days after radiation stay in the farm 18 days) presented normal cristae albeit compressed by the dilated perilymphatic space normal maculae the papilla of Corti with exception of a few sections in normal condition Another animal (weight 205 g perfused 20 days after radiation with a total stay at the farm of 35 days) showed the cupulae compressed by perilymphatic edema but otherwise normal cristae normal maculae and normal organ of Corti at both sides at one side a slight sero fibrinous labyrinthitis and some blood in the cochlea The 3rd animal (weight 205 g perfused after 20 days after radiation with a total stay at the farm of 45 days) presented normal cristae and maculae some perilymphatic dilatation and normal organ of Corti Reissner's membrane kept its normal position in all three cases

Radiated with 800 r the two groups together included 30 rats Seven showed infection free ears Long stay in the farm accounting for this low ratio

In the Roswell group after 2 days with normal cristae and maculae Corti's organ was in all turns destroyed (with one ear disregarded because of technical shortcomings) In the Charles River group three animals of average weight 117 g died in anesthesia 13 days after radiation having spent 11 days at the farm Perilymphatic edema in one case compressed the cupula of the horizontal crista otherwise cristae and maculae were normal in two cases the organ of Corti was destroyed with the contours here and there still identifiable in the 3rd it was destroyed in the basal turn to approach normalcy as it ascended against the apical turn The membrane of Reissner was everywhere straight

Radiated with 900 r the Charles River group offered 10 rats Among the 20 ears 13 were free of infection The first three (average weight 55 7 g two spontaneously dead all on the 13th post radiation day to which six pre radiation days at the farm must be added) showed the following The vestibular end organs although somewhat loose in their structure were otherwise intact Corti's organ was destroyed with the stria breaking up, except in the perfused animal where it was intact upward from the middle turn—possibly preserved in the congealing secretions of the serous labyrinthitis manifest within the cochlear duct Five animals (average weight 74 g perfused 4 days after radiation, 10 days in all at the farm) showed normal end organs,

with the exception of one case where one ampullar cupula was compressed by perilymph edema. In one case Corti's organ was intact albeit—or because—buried in serous transudate; in another case it was found intact in spite of beginning organization of the inflammatory products in the middle ear.

Among four animals of the Charles River group radiated with 1000 r (average weight 103 g, perfused on the 4th post radiation day, 18 days in all at the farm) four of the eight ears were free of infection, except for a slight serous labyrinthitis in one of them on one side. The end organs were intact, in one instance with ampullar cupulae compressed by perilymphatic edema, which was present in a considerable degree in another animal, too. In the case with serous labyrinthitis Corti's organ was intact, embedded in the serous secretions. The mass of the transudate by contraction pulled up with it and away from the Corti papilla the tectorial membrane, thus ending cochlear function in this sector.

RADIATION EFFECT ACCORDING TO THE SINGLE ELEMENTS OF INFECTION-FREE HEARING ORGANS

Tympanic membrane. It is known that in the rat, even in presence of severe middle ear infection, this membrane is infrequently perforated, compared to human conditions. In the uninfected cases it preserved its paper thin aspect, and wrinkling and smooth tears could be considered artifacts. In some cases (Fig. 1) the Shrapnel section was deeply retracted, reaching the body of the large ossicles. As it did not come to a perforation this retraction remained without pathological consequences. The mucous membrane of the tympanic aspect participated in the condition of the mucosal lining of the middle ear in general.

Middle ear. This region, so important from the viewpoint of non experimental infections, showed in uninfected animals few signs of radiation damage. Two rats of the Charles River series had some free extravasates—in the epitympanum and in the obturator foramen on one side. As both were sacrificed by decapitation some penetration from the pharynx through the tube cannot be excluded, albeit no blood was present in the middle ear of decapitated controls. The mucous membrane was often engorged without extravasations. No edematous or bleeding tendency could be noted. It is interesting to compare these findings with those found in the inner ear. The tubal orifice was never occluded and full patency of the tube itself was the rule. The mucosal lining of the ossicles shared the general condition in the middle ear.

Internal ear. It is well established that, as in humans, in a large majority of the cases, infection of the middle ear does not involve the internal ear. Many more inner than middle ears in this group could have served the



Fig 1 HE × 500 500 r Retraction of Shrapnel membrane

HE hematoxylin eosin HM Heidenhain Mallory (for all figures)

tabulation except for the fact that such ears were considered only where both were free of infection

Vestibular part Perilymphatic edema was a very frequent finding compressing the endolymphatic space in spots to a narrow slit Beyond 400 500 r this was practically a *standard finding* *Ampullar cristae with their cupulae* turned out to be very resistant in a number of instances compression by perilymphatic edema resulted in deflection of the cupula (Fig 2), this being the only damage to the ampullar organ Doses around 1000 and 2000 r resulted often in destruction—crista and cupula disintegrating in equal manner But even high doses could leave the ampullar organ intact (Fig 3, at 1000 r) In the uninfected cases no hemorrhage was observed in the peri or endolymphatic spaces

The *cochlear part* offered very diversified pictures The organ of Corti could remain at 400 r, totally unaffected (Fig 4) The same could be seen in the plane of the reticular lamina (Fig 5) while the nervous content of the modiolus (Fig 6) was, in this instance undisturbed at 2000 r A similar modiolar content was common even with destroyed Corti organs, it may be that the short post radiation life did not permit proceeding to deeper damage But disintegration could already start at low doses, and it must be remembered that in the controls Corti s organ was less well preserved than the vestibular organs Early change as seen in the reticular lamina

Fig 2 HI, 200 1000 r Bending of cupular crista by perilymphatic edema



Fig 3 HI 400 1000 r Intact crista with cupula

Fig 4 HL 400 500 r Intact organ of Corti

[illegible]



FIG 6 HM ×200 2000 r Slight edema of modiolar content normal vessels



FIG 7 HE 400 1000 r Slight derangement in reticular lamina

Fig 8 HE 400 1 00 r Corti destroyed (by edematous pressure)

Fig 9 HM 40 1200 r Cochlea filled with blood

showed up in wavy distortion (Fig. 7). The vascular stria was found in every degree of disintegration.

Impact on the organ of Corti resulted in a picture corresponding to trauma or other injury. The flat empty basilar membrane seen at prolonged acoustic trauma was not frequent here; the characteristic condition was a "blown up" organ (Fig. 8) as if disrupted by edema (cf. Ruedi & Furrer 1946, Figs. 80 c, 81 c, 82 c). At the same time it would not be possible to assume hydropic condition, as the membrane of Reissner was without exception in its standard position. The tectorial membrane, much more resistant than the Corti papilla, was frequently thrown against the Reissner membrane by some centripetal effect within the cochlear duct.

In contrast to the middle ear, extravasates were present in not less than eight of the 28 infection free rats. In the cochlea the extravasation was restricted mostly to the tympanic scala, once it was localized in the vestibular scala, turning up twice within the cochlear duct. One case, with 1200 r, showed the heaviest damage: blood in all cochlear spaces (Fig. 9). Here, too, the content of the cochlear duct repeated its most frequent disposition: "blown up" Corti with tectorial pushed up to the Reissner membrane, the latter straight. A rarer occurrence was destruction of the contents of the cochlear duct by blood, leaving a bare basilar membrane.

ADDITIONAL EVIDENCE FURNISHED BY INFECTED CASES

Generally, only cases free of non-experimental infection should serve as the basis of final tabulation of results. However, valuable information can be obtained from infected cases by an appropriate procedure (described in detail, Kelemen 1958). Familiarity with the lesions, chronic or recent, permits the results of the actual experiment to be distinguished among those developed before the animal was exposed to the agent in question, or even among those developing during the period of the experiment but independently from the first effect. It is of great help to know how much time is needed before one or the other pathological picture take shape, e. g. a recent hemorrhage should be recognized as such among signs of organization evidently needing a longer period to develop.

The crucial question is: Can it be decided whether the massive middle ear infections were present prior to, or should they be considered as caused by the radiation. Careful comparison with the controls, and more instructively with other animals seen on many other occasions, is the guide in this difficult terrain.

The substance of the *tympanic membrane* was seen dissected by hemorrhage separating the skin from the mucosal layers (Fig. 10). Localized hemorrhagic extravasate created bullae (Figs. 11, 12) and polyps (Figs.



Fig 10 HM 40 500 r Tympanic membrane separation of layers by extravasate space formation

13 14) Cases with perforations offered the characteristic sign of infolding epithelium covering the edges of the perforation—one (Fig 15) after 1500 r and only one day another (Fig 16) after 300 r after 3 days Whether a similar state can develop in such a short time is questionable Examples of maximally retracted but still not perforated Shrapnel membranes were given above (Fig 1)

In one instance (Fig 17) with only 100 r but after 14 days a mass of epithelial lamellae found its way along an invagination of the tympanic membrane but did not penetrate the drum entirely as a cholesteatomatous mass This was the only case of its kind in the present series although under different experimental conditions—poisoning with arsenious acid (Kelemen Ambrus & Ambrus 1958)—extensive cholesteatoma formation was encountered Here the cholesteatoma remained restricted to the substance of the tympanic membrane 14 post radiation days were seemingly sufficient to produce this condition

It has been described (Ke 1958) how changes in the submucosa of the middle ear tell the story of the evolution of the pathological picture tree rings The underlying bone remained practically always intact Any hyperemia with periosteal hemorrhages (Fig 18) was obviously of recent origin and osteomyelitic lesions (Fig 19) of older origin the latter (500 r with 11

Fig. 11 HE, ×50,400 r. Tympanic membrane: hemorrhageous bulla.

Fig. 12 HE, ×50,100 r. Tympanic membrane. hemorrhageous bulla.

FIG 13 HM, ×40,300 r Polyp protruding through tympanic membrane

FIG 14 HL, ×50,100 r Prolapse of tympanic membrane

Fig. 15 HL ×100 1500 r Infolding of edges of tympanic perforation

Fig. 16 HI 2 00 r Infolding of edges of tympanic perforation



Fig 17 HE, × 50,100 r Cholesteatoma in (infiltrated) tympanic membrane

post and 6 pre radiation days at the farm) *with osteoblastic seams* characteristic of the frantic attempt to repair as well as of fistulization The findings were in accord with the description of Warren a zone of demarcation between the normal and irradiated bone is absent, one shading off into the other; there is no *sequestration in the ordinary sense* Otherwise even the most fragile parts of the ossicles remained intact amid organized, plus recently added *suppurative masses* Fresh blood (Fig 20) could be identified as of acute origin, while advanced organization (Figs. 21, 22) was unmistakably a chronic formation These cyst like "rosaries" date the process, according to Manasse (1917) a considerable period is needed before similar organization starts Any role of the radiation in their formation should be excluded

Secretions in the lumen of the middle ear often found a narrow split between the thickened walls (Fig 23), or the space was divided by organized masses—bands between tympanic membrane and promontory. The recently-produced mass proceeded to colliquation of the organized layers by penetrating the latter (Figs 10 and 11 of the 1958 publication) while attempts to reabsorb the still liquid mass (Fig 24) by resorption buds projected from the fibrotic, organized submucosa are signs of reversibility.

It was hard to build up a post-radiation picture on a basis of the *products*



Fig. 18. H.M. × 150 800 r. Periosteal extravasation (bulla wall)

of chronic and acute infection. It was different with *hemorrhages* which were not present in the controls and showed every sign of recent origin. Larger basal vessels of the submucosa and fine vascular network in the granulation tissue belong here (Fig. 13, 19, 58) but they still can be explained by the organization process, not so free bleeding that fills the middle ear lumen (Fig. 25) or the membrane of the round window as pressed inward (Fig. 26) with characteristic spilling of blood into the scala tympani of the basal turn. Hemorrhages appear even in the old organization products (Figs. 27, 28).

While in the middle ear an evaluation of the infectious products remained the main problem, the requirement was different in the *internal ear*. Propagation of infection from the middle to the inner ear is infrequent. 90 rats (180 ears) of the Roswell group showed a single one-sided labyrinthitis, while the Charles River group (55 rats with 110 ears) carried this condition practically always in the serous form, to only 10 ears. A consequence of the inner ear being free of infection was that other changes—agonal, post mortem, artificial—were left high and dry to be distinguished from radiation effects.

The next most remarkable fact is the discrepancy between the state of the vestibular and the cochlear end organs. With few exceptions, in the

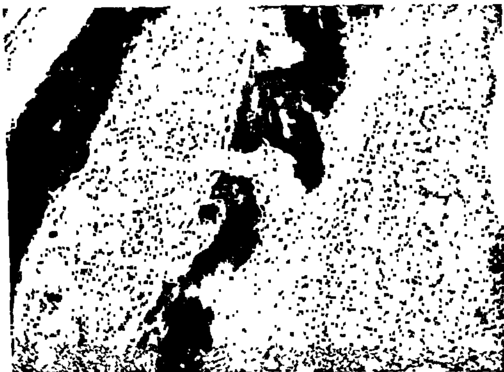


Fig 19 HE, × 100,500 r Osteomyelitis, bulla wall with fistula

vicinity of severely damaged Corti organs unharmed cristae and maculae were found

But even the organ of Corti frequently showed remarkable resistance. Some examples will be enumerated for the different end organs

The macula (Fig 29) shown here suffered only a slight edema, after 800 r, 10 post and 20 pre radiation days at the farm There were blood, scattered clumps of pus in both middle ears and, in the other ear, serous labyrinthitis A crista (Fig 3) with its cupula was unimpaired after 1000 r, 4 post and 14 pre radiation days at the farm Destruction begins with edematous loosening of the tissue of the crista (Fig 30), but the same vestibule contained an intact macula (Fig 29)

Depression of the cupula by perilymphatic edema (Fig 2) has already been mentioned The beginning of crista disintegration was seen amid serous labyrinthitis while, against perilymphatic engorgement and extravasation, the cupula was more resistant than the macula in its neighborhood, or the crista itself

An organ of Corti (Fig 31)—at 300 r, 3 post- and 18 pre radiation days at the farm—was edematous, with globular bodies characterizing a beginning degeneration and otherwise well differentiated with the hair of the internal and external hair cells clearly discernible, the structure of the tectorial was slightly edematous but clear Among the infected cases the "empty" basilar

FIG 23 HE, ×50, control Intact organ of Corti opposite organized mass at promontory

FIG 24 HE 50 control Resorption bud, from organized submucosa into fluid secretion

Fig. 25. HM ×50 800 r. Recent extravasate filling tympanic cavity

Fig. 26. HM 100 000 r. Extravasate in niche of round window [illegible]

FIG 27 HM × 50 800 r Hemorrhage in cystic organized mass in middle ear

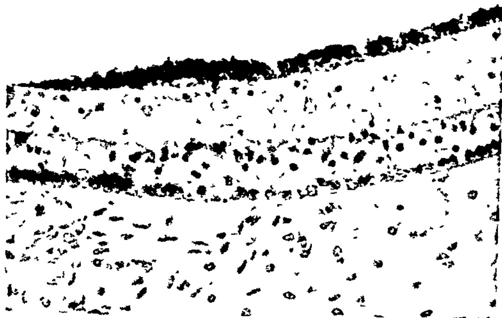


Fig. 29 HE, ×400,800 r Part of utricular macula intact

Fig. 30 HE ×400,800 r Edematous loosening of ampullar crista

Fig 35 HI ×200 900 r Within the cochlear duct complete destruction only tectorial preserved

Fig 36 HM 200 800 r Corti destroyed bleeding from stria tectorial intact under extravasate which reached Reissner's membrane

Fig 37 HM, ×500, 1000 r Corti, middle turn, intact opposite pus between promontory and ossicle



Fig 38 HI ... control Intact reticular lamina opposite organized mass at promontory, which breaks up the cochlear capsule

products harboring fresh pus, to penetrate the bone of the cochlear capsule The Corti organ was intact (Fig 37, 1000 r, 4 post and 14 pre radiation days at farm) while outside, at the promontory, hemorrhageous suppurative masses were piled over the engorged mucous membrane Henshaw (1958) explained that larger doses are required to produce detectable effects in the nervous system and that the responses occur at different times after the exposure

DISCUSSION

Radiation effect as the provoking pathological factor can be assessed only after taking into due consideration non experimental pathology, agonal, postmortem and artificial changes Hemorrhages stand out clearly, the more so as none of the effects of the enumerated extra experimental agents are characterized by extravasations Thielemann had already mentioned the bleeding tendency, so did Ivanov, Kozlov spoke of vascular damage, Zlotnikov reported labyrinthine hemorrhages, Borsanyi, Blanchard and Thorne assumed a transient vasculitis in the inner ear Our examinations detected extensive hemorrhages around the sensory organs of the inner ear The altered vessels, often inconspicuous in themselves, may bring about profound changes in those tissues dependent on them (Warren, 1944)

Regarding dosage, it is impossible to establish thresholds above which steady changes can be expected According to different regions—middle or inner ear or vestibular as against cochlear end organs or, again, vascular as against nervous structures—each element has to be judged for itself It is not possible to compose a paramount picture regarding the complete hearing organ Chilov learned this when he wanted to separate, by radiation, the action of the maculae from the action of the cristae for both kinds of end organs the present investigations showed identical, high resistance against x ray or cobalt

The period spent in the farm previous to radiation did not have any decisive influence although identical within one group, the pathology showed all kinds of variations There were remarkable episodes, as when a Charles River group, exposed to 1200 r spent 3 post and 34 pre radiation days in the farm four of the five animals remained free of infection, three showing hemorrhages in the inner ear Here, as in additional instances, radiation effect manifested by hemorrhages was present without infection, showing that one of the vascular effects of radiation, perilymphatic edema, was of non inflammatory origin

As in rats of numerous other experiments, middle ear infection was rarely followed by otitis interna The comparatively rare transmission of middle ear infection to the inner ear is so well studied in human pathology that discussion can be omitted here Direct exposure from the upper airways

through the tube to the middle ear and the comparatively sheltered location of the inner ear is one of the likely explanations The tube was always patent its secretions formed clear patches around its orifice within the suppurative mass in the middle ear

Hemorrhageous bullae of the tympanic membrane were seen several times (Fig 11 14) Only once did a cholesteatoma of the (much thickened) tympanic membrane develop but without break through to the middle ear lumen (Fig 17) Deep retraction of the Shrapnel membrane (Fig 1) did not reach the stage of fistulization

Very rarely did pus through a tympanic perforation pour into the external canal it is futile to rely on this sign for information regarding the presence of a middle ear process

The lining of the middle ear was frequently but not as a rule engorged—with or without extravasates Much more telling were foci of extravasate—in the old organized cystic tissue—witnesses that to the old infective agent a *new* destructive force has been added

The question of reversibility of the middle ear process can probably be answered as positive but in these experiments sufficient time was hardly given Even so although signs of reabsorption of suppurative masses were not lacking (Fig 24) they were observed only in controls (but see Kelemen 1958 Figs 10 11) It is impossible to state whether this tendency was counteracted by radiation

Tympanum and bulla were filled in different degrees by the secretions up to obliteration by organization products (Fig 23) A protective role of newly formed masses in the middle ear was sometimes assumed to cushion the effect of mechanical injury to the inner ear This factor can be left outside consideration here as the rays attacked from all directions

The question of whether radiation could have been the cause of suppuration must be denied Only after a longer period could cell debris cause a secondary suppuration Lack of new (reticular) bone as demonstrated in guinea pigs by Friedmann (1955) after experimental infections can be explained by different organisms and by the short post radiation periods In former experiments with arsenious acid poisoning (Kelemen Ambrus & Ambrus) *new bone in exostotic form* was demonstrated along the *tympanic* wall (Fig 4)

In the inner ear to hemorrhage is the most conspicuous radiation effect another circulatory lesion edema was added In the middle ear it manifested *itself merely in the tympanic membrane* resulting in space *formation* for example In the inner ear its main localization was in the perilymphatic spaces of the vestibule with compression of the endolymphatic space to a narrow cleft The bending of the ampullar cupulae by the displaced membranous wall was one *of the effects* Reversibility *of the edematous* condition in experiments of longer duration is conceivable

Observation of the organ of Corti offered two remarkable points among others One regards the numerous instances where an intact organ was

present—as contrasted in the middle ear at the opposite side of the cochlear capsule to severest acute and chronic lesions Even with knowledge of the comparatively rare invasion of the internal ear by middle ear inflammation this finding remains surprising The other point is the almost regularly observed different state of the organ in the different cochlear turns This was so frequent that derivation of the picture by different planes of sectioning could be excluded In the basal turn the papilla was flat compressed to change gradually to a profile well differentiated in the middle and intact in the apical turn Fernandez thought compression of the organ of Corti so that no structure can be recognized to be an artifact produced by some error in the histological technique but that this error should be confined to the basal turn is improbable A possible explanation at least for the cases with infected middle ears would be that in proximity of the round window the Corti organ is more exposed to damage blood or inflammatory products in the scala tympani near the secondary tympanic membrane are well known and frequent findings

Where Corti s organ was destroyed the autolytic pictures many times described were reproduced Some peculiarities deserve to be mentioned especially the many instances with Corti in destruction next to intact vestibular sensory organs Corti plus vestibular organs are generally mentioned as suffering identical fates under post mortem factors Fernandez explained that The autolysis changes in the vestibular receptors followed the same general pattern as that described for the cochlear receptors If this is so it can mean more than theorizing if one assumes that selective damage to the organ of Corti is an effect of radiation

Disintegration here presented a picture of edematous disruption leaving behind in several cases an empty basilar membrane The latter was practically never damaged On the other hand the old experience that the organ of Corti can be intravitally embedded during coagulation of a surrounding serous or fibrinous content of the cochlear duct was substantiated by perfect preservation But the state in which the organ was encountered followed no hard rules e g in a few animals found dead in the morning and fixation consequently inevitably delayed—preservation of the sensory organs was entirely satisfactory

Edema overproduction of fluid with pressure exerted from the direction of the basal and the lateral wall of the cochlear duct may have been the force pushing the tectorial membrane up all the way to the membrane of Reissner But the state of the latter—without a single exception in standard position—contradicts the presence of a similar flow Overproduction of fluid could have compressed the organ of Corti again this effect could not remain restricted to the basal turn

The tectorial in its rise to the Reissner membrane (Fig 33) seemed to pull the organ of Corti with it possibly initiating its disintegration The same effect was exerted by a shrinking sero fibrinous transudate Mygind (1945) discussed elevation of the tectorial membrane by a bulging membrane of

Reissner in connection with phenomena around the fixation For the present instances—with straight Reissner—it is simpler to assume that shrinking bands disrupt the tenuous attachment to the Corti papilla Several times the tectorial was intact over scant remnants of the organ of Corti This is in agreement with Fernandez who did not see dissolution of tectorial membrane left at room temperature—even 20 hours after death, when the organ of Corti was already fully disintegrated

Contact with even massive extravasates left the tectorial intact (Fig 36) Blood was found (Kelemen, 1963) to act as a specific poison to the substance of the cupulae This is another instance which warns against the too far reaching identification sometimes made, of the tissue of the tectorial with that of the cupulae

The vascular stria generally paralleled the condition of the organ of Corti Its disintegration was frequent, with extravasation from its surface

The modiolar contents showed considerable resistance with its compartments well filled (Fig 6) even in specimens with middle ear plus inner ear damage Slight edema was all that could be recognized it cannot be disclaimed however, that special stains would have demonstrated finer damage to the ganglionic tissue According to Warren, the neuron is one of the most resistant cells of the body

The nervous tissue in the internal acoustic meatus was often affected by the removal of the specimen and, after decapitation the canal may contain blood which, on the other hand when present can easily be flushed out To avoid faulty interpretation this area was omitted from the reporting

In a number of cases the smallest doses of anesthetics—many times applied without mishap in other experiments by the same hands—turned out to be lethal Whether radiation is followed by lowering of resistance against anesthetics must be decided by studies on a more general basis than the present

As to the findings of previous investigators reports regarding hemorrhages can be fully sustained Conclusion is different with regard to the descriptions of the state of the sensory end organs In some instances sharply defined characterization was given as to the effect of the particular dosage and of the time elapsed after radiation Post mortem changes were sometimes mentioned but pre experimental pathology practically never, or, in rare instances where mentioned they were disregarded Consequently every tabulation must be considered as being of questionable value and not acceptable for a comparative evaluation

SUMMARY

Young rats were exposed to full body radiation between 100 and 3000 r, 85 by x ray and 45 by cobalt. 15 served as controls. Sectional series of celloidin embedded temporal bones were examined under the microscope. To avoid the influence of non experimental pathology (a factor of great consequence) the findings were tabulated on basis of the infection free material with some data utilized (outside the tabulation) from cases showing disease independent from the experiment.

Hemorrhage stood out clearly as the leading damage, primary by direct acute extravasation and secondary by damaged vessels giving way. Tympanic membrane, mucous membrane lining of the *middle ear* inclusive of the ossicles showed engorgement with extravasation. Sometimes the bone suffered by participation of the periosteum in the general process. The *inner ear* was conspicuous by remaining in many instances intact next to a heavily damaged tympanic cavity. Damage here manifested itself in vascular phenomena, perilymphatic edema with compression of the endolymphatic space in the vestibular part and destruction of the elements of the cochlear duct with exception of the remarkably resistant tectorial membrane by hemorrhage from the stria vascularis. The basilar and the Reissner membranes remained intact, the latter without exception in standard position. Most conspicuous was destruction of the papilla of Corti and surrounding elements in its vicinity while cristae, inclusive of their cupulae, and maculae proved to be practically immune to radiation in the here applied dosage. On the other hand the organ of Corti, while in a number of instances destroyed next to intact vestibular end organs, was in other cases intact next to massive infection in the adjacent middle ear. In different cochlear turns the organ of Corti could show different degrees of disintegration, the apical turn generally containing the organ with least damage.

Reports of previous investigators could be fully sustained as to hemorrhages, while statements regarding the sensory end organs, as non experimental pathology remained outside of consideration, were inacceptable for comparative evaluation.

REFERENCES

Berberich J, 1936 Radium Roentgenschaedigung des Trommelfelles *Folia oto-laryngol orient* 2, no 3-4, 323-324

Berberich, J, and Kelemen, G, 1938 Erkrankungen des Ohres In Cohrs, P Jaffé, R, Meessen, H, (eds) *Pathologie der Laboratoriumstiere*, vol 1, pp 671 698 Springer Verlag, Berlin Gottingen Heidelberg

Berg, N O, and Lindgren, M, 1961 Dose factors and morphology of delayed radiation lesions of the internal and middle ear in rabbits *Acta radiol* 56, no 4, 305-319, Feb

Block, E, 1952 Rontgenschadigung des Schlaefenbeines *HNO 3* no 2 45-46 Feb

Borsanyi, S, Blanchard, C L, and Thorne, B, 1961 The effect of ionizing radiation on the ear *Annals Otol, Rhinol & Laryngol*, 70, no 1, 255-262, March

Chilov, K, 1927 Über die Anwendung von Radiumemanation in der experimentellen Labyrinthologie *Monatsschr Ohrenheilk & Laryng Rhinol* 61, no 7, 768-794, July

Clemensson, C J, and Nelson A, 1960 Sense organs In Errera M, and Forssberg, A (eds) *Mechanisms in Radiobiology*, vol 2 p 145 Academic Press, New York & London

Desjardins, A U, 1931 Action of Roentgen rays and radium on the eye and ear *Am J Roentgenol*, 26, no 6, Dec, sect 3 921-942

Ewald, C. A, 1905 Die Wirkung des Radium auf das Labyrinth *Zentralbl f Physiol*, 19 no 10, 298-299, Aug 12

Fernandez F, 1958 Postmortem changes in the vestibular and cochlear receptors (guinea pig) *Arch Otolaryngol*, 68, no 4 460-487 Oct

Friedmann, I, 1955 The comparative pathology of otitis media-experimental and human *J Laryngol & Otol*, 69 no 1 27-50 Jan and no 9 588 601 Sept

Gerstner, H B, Konecci E B and Taylor W F 1954 The effect of local brain irradiation on the pinna reflex of the guinea pig *Rad Res* 1 no 3 262 269 June

Girden, E 1935 Effect of Roentgen rays upon hearing in dogs *J Comp Psych*, 20, no 2, 263-290 Oct

Girden, E and Culler E 1933 1934 Auditory effects of Roentgen rays in dogs *Am J Roentgenol* 30 no 2 215 220 Aug and 32 no 5 675-679 Nov

Guggenheim P Clements L Schlesinger A Peartree P Jurgensen W, and McDonald R 1961 Effects of x ray on the tympanal mesenchyme cycle *Arch Otolaryngol*, 74, no 2 185 188 Aug

Gyorkey J and Pollock F J 1960 Radiation necrosis of the ossicles *Arch Otolaryngol*, 71, no 5 793-96 May

Henshaw P S 1958 Whole body irradiation syndrome In Claus W D (ed) *Radiation Biology and Medicine* pp 317 340 Addison Wesley Publishing Co Reading Mass

Hertwig P 1944 Die Genese der Hirn und Gehoerorganmissbildungen bei roentgenmutierten Kreisler Maeusen *Zeitschr f menschl Vererbungs u Konstitutionslehre* 28 no 2 327 354, May

Hicks S P 1953 Developmental malformations produced by radiation *Am J Roentgenol, Radium Ther & Nucl Med* 69 no 2 272 293 Feb

Ivanov M I 1957 Changes in the internal ear of animals following x ray irradiation *Vestn ORL* 20 no 5 78-83 Sept Oct

Kelemen G 1955 Experimental defects in the ear and the upper airways induced by radiation. *Arch Otolaryngol* 61 no 4 405-418 April

1958 Otitic reinfection An experimental study *Arch Otolaryngol*, 67, no 3 257 267, March

— 1960. Germfree reared animals in otolaryngic experimentation. *Proc 2 Sympos Gnotobiotic Technol*, Univ Notre Dame, Ind, 1960, 183–185

— 1962. Histology of nasal and paranasal cavities of germfree reared and ex germfree rats *Acta Anatom*, *48*, no 1–2, 108–113

— 1963 Hemorrhage A specific poison to tissue of ampullar cupulae *Arch Otolaryngol*, *77*, no 4, 365–375, April

Kelemen, G, Ambrus, J L, and Ambrus, C M, 1958 Arsenious acid and the hearing organ *Acta Oto Laryngol*, *49*, no 1, 53 63, Jan Feb

Kelemen, G, and Sargent, F, Jr, 1946 Non experimental nasal pathology in laboratory rats *Arch Otolaryngol*, *44*, no 7, 24–42, July

Kozlov, M J, 1959 Changes in the peripheric section of the auditory analyzer in acute radiation sickness *Vestn ORL*, *21*, no 2, 29–35, March–April

Manasse, P, 1917 *Handbuch der Pathologie des menschlichen Ohres* J F Bergmann, Wiesbaden

Marx, H, 1909 Untersuchungen über experimentelle Schädigungen des Gehororganes B Über Schädigungen des Gehororganes durch Strahlenwirkung *Zeitschr f Ohrenheilk*, *59*, no 1, 192–210, July

Miani, P, 1959 Sul comportamento delle cilia vibratili della tuba di Eustachio e della trachea sotto l azione dei raggi x e dei raggi gamma del radium *Minerva ORL*, *9*, no 5, 143–168, May

Miani, P, and Margucci, L, 1960 L'azione dei raggi beta e gamma dell 198 Au sulla attività ciliare *Minerva ORL*, *10*, no 12, 341 344, Dec

Moskovskaya, N V, 1959 Influence of ionizing radiation on the function of the vestibular analyzer *Vestn ORL* *21*, no 4, 59 62, July–Aug

Murphy, J E, and Harris, J D, 1961 Negligible effects of x radiation of the head upon hearing in the rat *J Aud Res*, *1*, no 2 117 132, Jan

Mygind, S H, 1945 Experimental histological studies on the labyrinth 3 The endolymphatic compression *Acta Oto-Laryngol*, *33* no 2 86 116, March April

Novotny, O, 1951 Sull azione dei raggi x sulla chiocciola della cavia *Arch Ital Otol, Rinol, Laringol*, *62*, no 1, 15 19 Jan Feb

Nylen, C O Engfeldt, B, and Larsson, B, 1960 The effect of local irradiation of the labyrinth in the rat with ionizing particles *Acta Oto-Laryng* suppl *158* 217 218, June

Pautov, N A, 1958 Healing of operative wounds of otolaryngeal organs in animals subjected to x ray radiation *Vestn ORL*, *20* no 5 14 19 Sept Oct

— 1959 Treatment of operative wounds of ENT organs in animals subjected to x ray irradiation *Vestn ORL*, *21* no 4 30–35, July Aug

Ruedi L and Furrer, W, 1946 Das akustische Trauma *Practica Oto Rhino-Laryngol* *8*, no 4 177 372 July Aug

Schuknecht H F 1953 Techniques for study of cochlear function and pathology in experimental animals *Arch Otolaryngol*, *58* no 4, 377–397 Oct

Thielemann M 1928 Experimentelle Roentgenschaedigung des Ohres *Fortschr a d Geb Roentgenstr* *37* no 4 563 564 April

Warren S 1944 The histopathology of radiation lesions *Physiol Rev* *24*, no 2 225 238, April

— 1958 Radiation injury In Harrison T R (ed) *Principles of Internal Medicine*, pp 797 803 McGraw Hill Book Co, Inc New York Toronto London

Work W P 1950 Lesions of the external auditory canal *Annals Otol, Rinol, Laringol*, *59*, no 4 1062 1087 Dec

Zlotnikov S A 1961 Pathohistologic and morphologic changes of the vestibular analyzer due to radium emanation *Vestn ORL* *23* no 2 19 23, March April

*

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1962

Acta

OTO-LARYNGOLOGICA

S U P P L E M E N T U M 186

FUNCTIONAL CORRELATIONS BETWEEN THE DIENCEPHALIC NYSTAGMOGENIC AREA (DNA) AND THE VESTIBULAR NYSTAGMOGENIC AREA (VNA)

BY

PIERRE MONTANDON

(GENEVA)

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

★

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1963

From the Physiological Institute, University of Basel (Switzerland)
(Director, Prof M Monnier)
and from the Oto-rhino-laryngologic Clinic, University of Geneva (Switzerland)
(Director Prof A Montandon)

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 186

FUNCTIONAL CORRELATIONS BETWEEN THE DIENCEPHALIC NYSTAGMOGENIC AREA (DNA) AND THE VESTIBULAR NYSTAGMOGENIC AREA (VNA)

BY

PIERRE MONTANDON

(GENEVA)

UPPSALA 1964

PRINTED IN SWEDEN BY
Almqvist & Wiksells
BOKTRYCKERI AKTIEBOLAG
UPPSALA 1964

My thanks are due to Professor M Monnier, Director of the Basel Institute of Physiology, who inspired and directed this thesis, to Professor A Montandon, Director of the Geneva University Oto Rhino Laryngology Clinic, who kindly placed at our disposal his electronystagmography laboratory for the rotatory vestibular stimulation of our animals and to Mrs Libois who helped us with this I should also like to thank Mr S Graber for his valuable contribution to the iconography of this work, Mr Robert Bertrand, M D, from Montreal, who kindly did the translation into English, and Mrs P Montandon who carried out the histological controls

I INTRODUCTION

In 1958 Lachmann Bergmann and Monnier using the stereotaxic method of Monnier and Gangloff described a mesodiencephalic nystagmogenic area in the rabbit composed of structures medial to the reticular thalamic nucleus and the lateral geniculate body

The electrical stimulation of this area produced both an ocular and cephalic nystagmus in a horizontal plane with slow and rapid components The rapid component which indicates the direction of nystagmus was contralateral to the mesodiencephalic stimulation

Lachmann Bergmann Weinmann & Welner (1958) further studied the relationship of this central nystagmus to the nystagmus produced by the stimulation of the vestibular receptors and considered the possibility of two different mechanisms acting upon a common substratum

In 1959 Bergmann Lachmann Monnier and Krupp employing frontal and longitudinal sections of the brain stem came to the following conclusions

1 The afferent inhibitor impulses of cortical and retinal origin moderate both the central and the vestibular nystagmus

2 The efferent pathways of the diencephalic nystagmogenic area proceed caudally towards the oculomotor nuclei

3 An interruption of these pathways abolishes the central nystagmus but does not modify the vestibulo ocular reflex arc in a marked way

4 The section of the median pathways between the bulbo protuberantial and the mesencephalic region abolishes the vestibular nystagmus it also modifies the central nystagmus (elevation of the threshold and disruption of the period of latency and after discharge)

5 Frontal sections in the medial part of the mesencephalon in the region of the oculomotor nuclei sometimes abolish the vestibular nystagmus sometimes the central nystagmus sometimes both

6 The integrity of the pathways crossing the midline is indispensable for the production of all kinds of nystagmus

7 Longitudinal sections extended anterior to the oculomotor nuclei produce a spontaneous temporary dissociated and divergent (horizontal or vertical) nystagmus which the authors attribute to an irritative lesion of the posterior commissure

It seemed interesting for us to further the above experiments on the diencephal nystagmogenic area with a series of experiments with the purpose of elaborating the following

— Differ ted localization of the diencephalic nystagmogenic area

Compar f the characteristics of the diencephalic nystagmus to

those of the nystagmus produced by rotatory stimulation of the vestibular receptors

— Analysis of the perrotatory nystagmus in relation to lesions of the dien cephalic nystagmogenic area
— Analysis of the results produced by stimulation and coagulation of the bulbar vestibular nuclei
— Anatomical and functional relations between the diencephalic nystagmogenic area and vestibular nystagmus

We purposefully limited our observations to the nystagmic reactions and strived to obtain adequate experimental conditions By means of electronystagmographic recording, we were able to analyze quantitatively the nystagmic responses, keeping the animal away from inhibitory factors such as light and noise

II. METHOD

We used for our experiments the technic and stereotaxic Atlas of Monnier & Gangloff (1961); this technic allowed us to stimulate precisely determined structures of the brain of a conscious rabbit and to register simultaneously the cerebral electrical activities. The animal was fixed in a hammock in as normal a position as possible. *It was subsequently placed in a soundproof cage of Faraday* which was capable of being totally darkened.

1. *Stimulation*

(a) *Electrical stimulation of the mesodiencephalon*

Under local anesthesia (2 cc 1 % novocaine) a plexiglass socklet is screwed on to the surface of the skull. This socklet is perforated in such a way as to allow the passage of electrodes every 1.5 mm. Electrode guides on the socklet serve as reference points and allow adjustment of the electrode holder as well as the position and depth of the electrodes.

The electrode holder consists of three steel needles of the type designed by W. R. Hess, 0.25 mm in diameter, insulated except at their extremities, 16 mm long and 1.5 mm apart. Their position and depth in the brain stem must be previously calculated by means of the coordinates of the stereotaxic Atlas.

The stimulation is usually bipolar, monopolar in a few cases with the indifferent electrode a large copper surface in the form of the bit of a bridle placed in the mouth.

The stimulator is a Grass Model S III stimulator which produces rectangular pulses of which the length, frequency and voltage can be modified.

The parameters of optimum stimulation are: frequency = 50 pulses/sec, duration = 0.5 millisec, intensity = 0.1 to 1.0 volt. The electrical thresholds of the responses are measured in volts. *An interval* of at least one minute separates each series of stimulations in order to avoid the phenomena of fatigue or adaptation. The duration of each series of stimulations is 30 sec at most.

(b) *Electrical stimulation of the bulbar vestibular nuclei*

The stereotaxic socklet possesses an additional posterior prolongation with a 30° angulation towards the lower part. The exposure of the skull, the positioning of the socklet and the perforation of the holes are performed under local anesthesia (2-3 cc Novocaine 1 %).

The electrode consists of two platinum needles, insulated except at their extremities, a [illegible] mm apart (diameter = 0.40 mm, length = 27 mm). We

did not use steel needles of a smaller diameter, because they are too flexible for the desired length and too easily deviated by petrosal bone promontories

The stimulation was usually monopolar, with an indifferent electrode in the mouth but bipolar in a few cases

The stimulator and the parameters of excitation are exactly the same as for the diencephalon

(c) *Clockwise and counterclockwise rotating stimulation of the peripheral vestibular apparatus*

These were done in the Nystagmography Laboratory of the E N T Clinic of the University of Geneva under the direction of Professor A Montandon and according to the method proposed by him in 1954 under the name of *liminal rotatory stimulation* The rotatory stimulation is produced by a rotating table activated by an electric motor with hydraulic transmission on which are placed the subject to be examined and the recording apparatus The special rotatory stimulator for this research is an apparatus of very high precision capable of furnishing rigorous constant accelerations from the beginning to the end even for very small angular accelerations less than 1° per second per second The accuracy and the uniformity of acceleration of the turning table are directly controlled by an accelerorheometer (A Montandon & F Dittrich 1961) This procedure allows us to examine separately each horizontal semi circular canal It consists of three phases

1 A period of constant acceleration until an angular velocity of 90° per second is obtained

2 A period of rotation at a constant speed of 90° per sec for approximately three min

3 A period of constant deceleration of the same intensity as the acceleration until a complete stop

In every instance the stimulation is done both in clockwise and counter clockwise directions

The intensity of the stimulation is measured by the degree of angular acceleration and deceleration of the rotating table We submitted each animal to a series of increasing stimulations $1°/s^2$ $3°/s^2$, $6°/s^2$ $9°/s^2$ The deceleration of $1°/s^2$ was replaced by a sudden stop allowing the comparison of post rotatory nystagmus to perrotatory nystagmus

2 *Recording of Nystagmus* (Electronystagmography)

Ocular nystagmus can be recorded by the electronystagmographic technic described by M Monnier & H J Hufschmidt (1951) based on the reports of Meyers (1929) Mowrer Ruch & Miller (1936) Jung (1936) Leksell (1939) Jung & Tonnies (1948) This technic proved itself suitable for the recording of perrotatory vestibular nystagmus in the rabbit [A Montandon & M Monnier (1951) A Montandon M Monnier, A Croci & W Brunner (1952)

M Monnier & H Laue (1952)], as well as in human subjects [A Montandon & M Monnier (1953), A Montandon *et al* (1953–1962)] To each position of the ocular globe corresponds a certain distribution of the periorbital electro static potentials Any movement of the ocular globe will modify this distribution by displacement of the electrical corneo retineal axis The recording of these periorbital potentials is done in the following manner two silver electrodes are fixed to the skin by means of a conductive paste, one directly above the nasal angle of the palpebral fissure, the other directly below the temporal angle These two electrodes are in the same plane as horizontal nystagmic movements For the study of vertical and rotatory nystagmic movements, a second bipolar derivation was used, using the temporal electrode and a third electrode placed above the temporal angle The potentials derived from these electrodes were registered on an electroencephalograph having four channels Lateral eyeball movements to the right produced upward electrographic excursions, while those to the left, downwards, according to the convention usually used (A Franceschetti, M Monnier, A Montandon & P Dieterle, 1959)

To record the nystagmus produced by electrical stimulation of the diencephalon and vestibular nuclei, a 16 channel Schwarzer electroencephalograph was used, allowing simultaneous amplification with different time constants (0 1, 0 3, 0 7, 1 0)

In order to determine the optimum time constant, nystagmographic controls were made on a cathode ray oscilloscope (Tectronix, Type 502) The DC amplifier gives an immediate reproduction of the slow and rapid movements of the eyeball, but has the disadvantage of producing non controllable errors On the contrary the AC amplifier gives, due to the time constant, an automatic return of the writing pen to a stable isoelectric line, a reference system which allows the correction of possible errors Recordings made with the usual amplifiers (coupling capacity, resistance) assembled in circuits with a time constant of 0 3 sec are essentially the same as those obtained with a DC amplifier when stabilized A longer time constant does not offer any advantage We can distinguish on a cathode ray oscilloscope, with direct coupling, an initial slow movement of the eyeball, immediately preceding the appearance of rhythmic nystagmus This initial movement classically termed primary slow component of nystagmus ", is not visible on tracings recorded on paper

As a rule we used mono ocular recordings (right eye) of the animal in complete darkness in order to eliminate the influence of light stimuli This experiment was always completed by direct observation of nystagmus and free behaviour of the animal In some cases the responses were filmed during stimulation

3 *Analysis of Nystagmographs*

The qu[illegible]tive analysis of the nystagmographic recording must take into considerati[illegible] following parameters

1 The intensity thresholds of the stimulus expressed in volts and degrees of angular acceleration

2 The intensity of the nystagmic response measured by the nystagmic frequency The frequency seemed to us the most consistently proportional criterion in relation to the stimulus intensity On nystagmographs produced by stimulation of the diencephalic and vestibular centers we counted the number of nystagmic beats between the 10th and 20th second of stimulation On the nystagmographs of rotatory origin we considered the mean frequency of the nystagmic beats immediately after the onset of the nystagmus

3 Finally we measured the latency of the first fast component of the nystagmus and also the duration of the after discharge at the end of the stimulation Only in certain particular cases was the relative amplitude of deflections calibrated in microvolts taken into consideration

4 *Stereotaxic and Anatomic Localisation of Stimulated Points*

The stereotaxic technic and the recent Atlas of Monnier & Gangloff (1961) allowed us to have a precise topographic localization of all stimulated points At the end of each experiment we coagulated the point having responded to an electrical stimulation of minimum voltage and after a few days the animal was killed and its brain was removed for histological control of the coagulated points (according to the Loyez Weil method staining of the myelin fibers)

5 *Analysis of the Nystagmic Response to Rotatory Stimuli after Coagulation of the Diencephalic Nystagmogenic Area (DNA)*

The nystagmic responses to the rotatory stimulation were elicited before and after unilateral destruction of the diencephalic area After recording a control vestibular response the right or left nystagmic area was coagulated by the high frequency Wyss coagulator (1945) This instrument produces a high frequency current (500 000 cps) of controllable amperage and duration (35 milliamp for 10 15 sec) The effect of the lesion was always checked by a stimulation at 5 volts

During the 10 to 15 days following the lesion the nystagmic response of the peripheral receptors to rotatory stimulation was recorded daily at the E N T Clinic of Geneva by liminal rotatory stimulation as described by A Montandon with angular accelerations of 1° 3° 6° and 9°/s² in both directions The animals were then sacrificed and the lesions of the DNA histologically examined

6 *Histological Study of Degeneration Secondary to Lesions of the Diencephalic Nystagmogenic Area (DNA)*

On certain animals secondary degeneration of efferent projections of the DNA was the object of histological study according to the technic of Marchi (osmic acid stain of myelin degeneration) A perfusion of the brain

with potassium bichromate was done immediately after death. Even though the impregnation of degenerated structures is not as marked for the rabbit as for the cat or the monkey, we were able to notice well defined degeneration.

7. *Analysis of Functional Correlations between the Diencephalic Nystagmogenic Area (DNA) and the Vestibular Nystagmogenic area (VNA)*

We stimulated simultaneously the DNA and the VNA with the same parameters (50 impulses per sec of 0.5 millisec duration and 0.5 to 1.0 volt intensity) by two unsynchronized Grass stimulators.

The stimulation of the vestibular nystagmogenic area was of 30 sec duration. *From the tenth to the twentieth sec by means of a second stimulator,* the ipsilateral diencephalic nystagmogenic area was simultaneously stimulated. The same procedure was repeated with stimulation of the contralateral diencephalic nystagmogenic area. Conversely, during stimulation of the diencephalic nystagmogenic area, the vestibular nystagmogenic area was stimulated for 10 sec. This procedure was repeated many times. The frequency and amplitude of the nystagmic movements were recorded before, during and after the superimposition of the stimulations.

III. RESULTS

1. *Localization of the Diencephalic Nystagmogenic Area (DNA)*

On 11 rabbits the diencephalon and the mesencephalon were stimulated with the following parameters: frequency = 50 c/s, duration = 0.5 ms, intensity = 0.1 to 1.0 volt. We noted at least two or three different points 1.5 mm distant from each other, which produced nystagmic response. The further we went from these points, the higher the threshold voltage becomes and it is accompanied by secondary reactions (agitation, cries, convulsions). In order to obtain a discrete localization, it is important not to include responses produced by an intensity higher than 1 volt.

(a) *Analysis of responses* (Fig. 1, Table I)

After a latency period of 1 to 10 sec. the nystagmic response seems to start with a slow and ipsiversive deviation of both eyes (towards the stimulated side) followed by a sudden movement in the other direction, which constitutes the initial fast component of the nystagmus. The fast component of the nystagmus is toward the side opposite the stimulated side; it is called "contraversive". Its maximum frequency is progressively attained in about 10 sec. and maintains itself at the same rate even if the stimulation is supraliminal. If the stimulus becomes infraliminal, the nystagmus slows down and disappears before the end of the stimulation.

The rate of nystagmus in the rabbit can be very slow while remaining regular: regular nystagmic movements can be observed at a frequency of 2 per 10 sec. Also, as the stimulus intensity is increased, the nystagmic frequency increases, but never exceeds 5 beats per sec.; usually, it attains a maximum of 2 to 3 beats per sec. which cannot be increased, whatever the stimulus intensity.

The nystagmus amplitude varies little during stimulation; it is not proportional to the stimulus intensity, nor does it have any constant relationship to the nystagmus frequency.

Following supraliminal stimulation, there exists a nystagmic after-discharge in the same direction, with little or no variation of amplitude, and a gradual diminution of frequency. The after-discharge lasts on the average from 2 to 10 sec.

The nystagmus was visually controlled on all animals and filmed in certain cases. This control, done in the presence of light, required a stimulation intensity two to three times greater than when in darkness. The behavior of the "free" animal was observed and filmed. The ocular nystagmus was frequently accompanied by a cephalic nystagmus in the same direction.

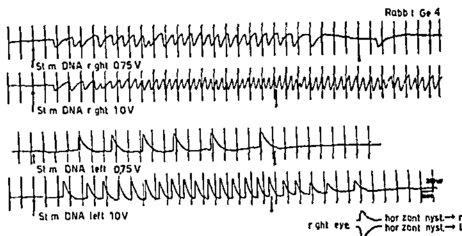


FIG 1 Electrical stimulation of the diencephalic nystagmogenic areas (DNA) right and left. At 0.75 volt the stimulation is liminal, the nystagmus is slow, it tends to slow down before the end of the stimulation. There is no post discharge. At 1.0 volt the stimulation is supraliminal, the nystagmus is more rapid and presents a post discharge.

synchronous with the nictitating membranes, mydriasis and exophthalmus. We never noticed any circus movements, the animal can come and go in all directions in a normal way.

(b) *Stereotaxic localization of stimulated points* (Fig. 2)

We transcribed all the stimulated points on the stereotaxic charts of the Atlas of Monnier & Gangloff (1961). Those for which the nystagmogenic points have a stimulation intensity threshold less than 1 volt confirm the experimental results of Lachmann, Bergmann & Monnier (1958). They are present in the following regions.

In the oral diencephalic segment: a fiber zone lateral to the reticular nuclei of the thalamus in the vicinity of capsula interna.

In the intermediate diencephalic segment (Fig. 2C–D, D): the lateral geniculate body, the latero dorsal nuclei of the thalamus, including the intralaminary system, the ventral nuclei of the thalamus and the optic radiation.

In the caudal diencephalic segment (Fig. 2 D, 1 L): lamina medullaris posterior. The intralaminary system between the pulvinar thalami, the lateral geniculate body and the medial geniculate body.

Oral mesencephalic segment (Fig. 2 F): the caudal part of the pulvinar thalami and the colliculus superior.

We did not obtain any nystagmic response in the caudate nuclei, the globus pallidus, the putamen, the medial region of the thalamus, the ventrolateral thalamus, the medial geniculate body, the nuclei of the posterior commissure or structures of the midline.

In r... ds to the stereotaxic method, the greatest number of positive points is situat... the planes be... D and DE at a depth of 10.5 mm from the inferior surfa... he socklet (Fig. 4D and DL).

TABLE 1

Rabbit	No. of stimulated points	Stimulation DNA: Stereotaxic localisation, optimal point	Stimulation DNA: Deep mm	Stimulation DNA: Intens volt	Nystagmic reponse: Direction	Nystagmic reponse: Latency/sec	Nystagmic reponse: Freq/min	Nystagmic reponse: Amplit microvolt	Nystagmic reponse: After discharge	Coagulation of the right DNA Nystagmic effect
755	48	R bc CD-DL	12	1.0	L	1	156	600	4	Nyst. to right (13/min 200 µV)
759	39	R bc D-DL	12	0.5	L	3	12	200	0	—
		L b D-DL	12	0,75	R	2	18	200	1	
761	42	R b L-LF	10.5	0.3	L	3	30	200	3	Nyst. to right (18/min 300 µV)
		R b D-DL	10.5	0.75	L	2	30	300	0	
772	81	R CD-D	10	0.5	L	5	18	250	0	
		L D-DE	10.5	1.0	R	6	24	600	0	
774	48	R b CD-D	10	0.5	L	4	36	150	3	—
		DE b-bc	10.5	0.5	L	4	18	150	0	
			10.5	1.0	L	3	126	300	15	
		L b D-DL	10	0.5	R	2	96	150	1	
780	87	R b CD-D	10.5	0.5	L	9	12	300	0	Died immediately
		c CD D	10.5	0.5	L	3	18	300	0	
		L c CD-D	11.5	0.5	R	1.5	18	150	2	
777	96	R b D-DE	10.5	0.75	L	4	42	350	16	—
		L D c-d	12	0.75	R	8	18	500	0	
787	66	R b L-LI	10.5	0.5	L	2	126	550	16	Nyst. to right (12/min 400 µV)
			10.5	0.25	L	3	48	400	0	
		L b D-DL	9	0.5	R	6	12	200	0	
790	57	R c CD	12	0.15	L	5	24	500	0	—
		L bd D	12.5	0.75	R	3	60	500	19	
801	51	R bc DI	12	0.15	L	3	66	150	0	Nyst. to right (6/min 200 µV)
806	21	R bc DI	10.5	0.5	L	5	24	150	5	—
		L bc D DI	10.5	0.5	R	5	36	300	9	

This stereotaxic localization was then verified on 12 rabbits used for experimental localization of the vestibular nystagmogenic area and on whom the diencephalic nystagmogenic area was stimulated in the stereotaxic planes bc D and DI at a depth of 10.5 mm. The threshold was never above 1 volt.

On two animals (887 and 896) the occipital cortex, the temporo parietal region and the cerebral cortex were systematically explored by means of a specially built stereotaxic socklet with an anterior and two lateral extensions forming a 45° angle. Stimulation with the following parameters: frequency = 30–50 imp/s, intensity = 0.5–5 volts, duration = 0.5–2 msec, did not produce any nystagmic response.

With the same stereotaxic socklet and while preserving the integrity of

the venous sinuses we were able to stimulate the medial region and particularly the posterior commissure but we could not reproduce the divergent dissociated nystagmus induced by a lesion of the posterior commissure and attributed to irritation (Bergmann Lachmann Monnier, Krupp, 1959)

(c) *Coagulation and localization of the stimulated points* (Fig 2)

To end our study the points producing the best nystagmic response were coagulated The anatomical control by histological slides stained according to the Loyez Weil method (staining of myelin fibers) further confirmed that the points of maximum response were located in the intermediate and caudal segments of the diencephalon *in the intralaminary system between* the lateral geniculate body, the dorso lateral nuclei of the thalamus the pulvinar thalami and the median geniculate body and in the optic radiation

Coagulation performed to localize the optimum nystagmogenic point *sometimes produced disturbances in four cases there appeared a spontaneous* nystagmus towards the coagulated side and *in one case* sudden death

To summarize, the stimulation with low intensity (frequency = 50 cps *duration* = 0 5 *msec* intensity = 0 1 to 1 volt) *of a well defined region located* in the dorsal lateral and caudal part of the diencephalon always produces a contraversive nystagmus This diencephalic nystagmogenic area does not extend towards the cerebral cortex *Coagulation of this region tends to produce* an ipsiversive spontaneous nystagmus toward the side of the lesion

2 *Influence of the Diencephalic Nystagmogenic Area (DNA) upon Vestibular Rotatory Nystagmus*

(a) *Nystagmic reactions of the peripheral vestibular system to rotatory stimulation*

Five rabbits were submitted to liminal rotatory stimulation (LRS) of A Montandon and the characteristics of the per and post rotatory nystagmus were analyzed The threshold for the rabbit *determined by the intensity* of rotatory stimulation necessary to produce a regular nystagmus lasting throughout the *acceleration or deceleration* is situated *between* 1° *to* 3°/s² of angular acceleration

Analysis of the nystagmic reaction Let us recall that clockwise rotation (towards the right) produces nystagmus to the right during acceleration and to the left during deceleration Conversely counterclockwise rotation (towards the left) *produces nystagmus to the left during acceleration and to* [illegible] right during deceleration So that in accordance with Ewald's experi[illegible] *we can consider rotatory stimulation as producing an ipsiversive* horiz[illegible] nystagmus towards the receptors that are being stimulated

This [illegible] mus begins after a latency of 1 to 10 sec Its *frequency* does not change dur[illegible] he stimulation and is never above 5 beats per sec The

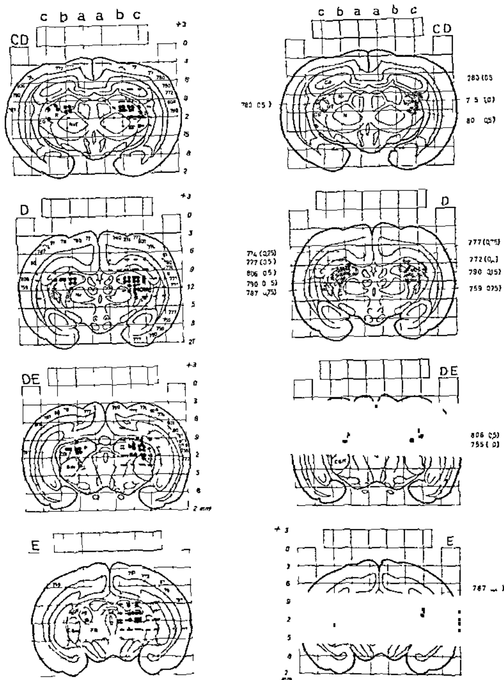


Fig. ? Diencephalic nystagmogenic area. Left *Stereotaxic localisation* of the diencephalic nystagmogenic area. The stimulated points are indicated on frontal sections of the rabbit's brain (Stereotaxic Atlas of Monnier & Gangloff 1961). Right *Histological localisation*. Lesions of optimal nystagmogenic points after high frequency coagulation (values between parentheses = nystagmus threshold in volt). CGL Corpus geniculatum laterale. CGm Corpus geniculatum mediale. CI Capsula interna. Cos Colliculus superior. CRa Corona radiata. FR Formatio reticularis. LMm Lamina medullaris medialis. LMP Lamina medullaris posterior. NLT Nucleus lateralis thalami. NR Nucleus reticularis. NVT Nucleus ventralis thalami. PT Pulvinar thalami. RO Radiatio optica.

minimum frequency is 1 beat per 10 sec (6 per min) With a smaller response, one obtains isolated beats at irregular intervals The amplitude does not present much variation and does not seem to have any relation to the intensity of the stimulation nor to the nystagmic frequency The perrotatory response is usually followed by a nystagmic after discharge in the same direction for 1 to 25 sec

The intensity of the responses, as measured by the frequency of nystagmic beats, increases with the stimulation There is no or a very small response at 1°/s², moderate at 3°/s², and very strong at 9°/s² Above 9°/s², its frequency and amplitude have a tendency of becoming irregular and frequently the animal becomes agitated For this reason, the most adequate intensity is 3°/s², which is close to the threshold

Accelerations and decelerations of the same intensity produce similar reactions

The post rotatory nystagmus, produced by a sudden stop from a constant speed (90°/s), recalls by its pattern the after discharge of supraliminal stimulation There is maximal nystagmic frequency and amplitude which gradually diminish, frequently the animal is agitated and the recording disturbed

Contralateral nystagmic stimulations usually produce similar and reproducible responses However, two animals (Ge 2 and 749), having an otitis, presented a slight asymmetry (directional preponderance)

To summarize, rotatory stimulation produces a horizontal nystagmus, preceded by a latency period, with a regular frequency proportional to the stimulus intensity, and followed by a nystagmic after discharge in the same direction The nystagmic threshold of the rabbit is situated between 1° and 3°/s² of angular acceleration or deceleration

(b) *Result of the DNA stimulation upon rotatory and calorically induced vestibular nystagmus*

Lachmann, Bergmann, Weinmann & Welner (1958), using as we did the stereotaxic method of Monnier and Gangloff and the rotatory stimulator of A Montandon demonstrated that the nystagmus produced by electric stimulation of the diencephalon can be activated or inhibited by rotatory stimulation of the peripheral vestibular apparatus If the left area is stimulated, a nystagmus to the right is obtained Counterclockwise rotation with a 3°/s² acceleration will inhibit and after a few seconds, completely mask the electrically induced nystagmus of the diencephalon When the rotation attains a speed of 90°/s, and is kept constant the diencephalic induced nystagmus reappears with the same frequency as before If we then give, by sudden [illegible]n of the rotation a new stimulation in the opposite direction the dience[illegible] [illegible] nystagmus is greatly accelerated

[illegible] thermal stimulation of the peripheral vestibular apparatus interferes with th[illegible] the stimulation of the diencephalic nystagmogenic area in the same ma[illegible]r

The activation of the nystagmus is manifested on increase in frequency and its inhibition by a masking of the reaction The nystagmographic recording is not disrupted by these competitive phenomena

Ether pentobarbital morphine and atropine modified in the same manner the diencephalic and vestibular nystagmus They cannot suppress one or the other of the nystagmic responses

To summarize peripheral vestibular stimulation enhances contralateral DVA electrical stimulation and inhibits ipsilateral DVA electrical stimulation

(c) *Result of DVA destruction upon rotatory induced nystagmus*

On six animals after studying the rotatory induced nystagmus by liminal rotatory stimulation (LRS) we stimulated and then destroyed the right or left DVA by an extensive coagulation In three cases this produced an ipsiversive spontaneous horizontal nystagmus It was never followed by paralysis circus movements or deviation phenomena In the days following the lesion the LRS demonstrated either a manifest or latent spontaneous nystagmus towards the affected side or an important directional preponderance of the nystagmus also towards the affected side

The manifest spontaneous nystagmus usually has a slow or moderate frequency It is accelerated during synergic rotatory stimulation It is masked by antagonistic rotatory stimulations This masking is usually preceded by a latency period and lasts 5 to 10 sec after the end of the stimulation Furthermore upon recurrence the spontaneous nystagmus is stronger than before

The latent spontaneous nystagmus is more or less easily detected sometimes by any stimulation (needle pinching) of the animal or by any rotatory stimulation sometimes only in the period following an antagonistic rotatory stimulation of strong intensity or a synergic rotatory stimulation It can sometimes persist as a spontaneous nystagmus during the entire test It can paradoxically be increased by antagonistic stimulations of small intensity We considered as *directional preponderance* any increase in the nystagmus frequency in a definite direction in relation to the other The difference usually remains the same at all degrees of acceleration except in the few cases where the preponderance reverses itself under the influence of high intensity stimulation (9°/s² or sudden stop at 90°/s)

The intensity of the directional preponderance varies from day to day sometimes being a simple preponderance sometimes a manifest or latent spontaneous nystagmus *From the fourth to the eighth day* the reactions seem to equalize Then there appears a preponderance in the other direction and on *the tenth day* following the DVA coagulation the nystagmic frequency towards the contralateral side of the lesion is faster than the one *towards* the side of the lesion

During the entire evolution of this post lesional picture the direction of the directional preponderance and the *variation of* nystagmic threshold are concordant (Figs 3 and 4)

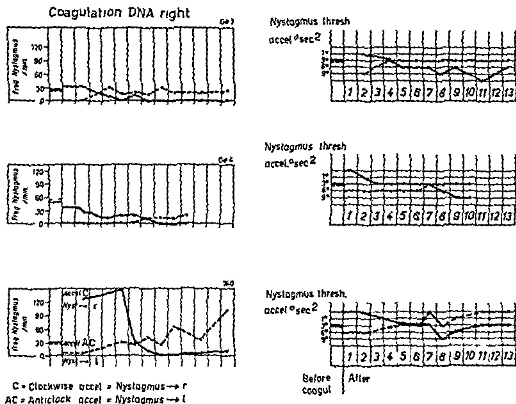


FIG 3 Action of coagulation of DNA on perrotatory nystagmus Coagulation of the right DNA of three rabbits (Ge1, Ge4, 740) Daily analysis of nystagmic frequency (clockwise acceleration) (C) and anticlockwise (AC) of 3°/s² and of the nystagmic thresholds of the liminal rotatory stimulation (intensity of acceleration necessary to produce a nystagmus of regular frequency) On this diagram Threshold below 1° = spontaneous nystagmus Threshold above 9° = nystagmus abolished In the three cases the coagulation produced a directional preponderance of the nystagmus towards the side of the lesion The frequency of the nystagmus beating towards the side of the lesion is higher than that of the nystagmus in the opposite direction Parallel variation of the nystagmic thresholds the threshold of the nystagmus beating towards the side of the lesion is lower than that of the nystagmus in the opposite direction After 3-7 days inversion of the directional preponderance

If the nystagmus to the left is predominant (in relation to the nystagmus to the right), the acceleration necessary to produce a nystagmus to the left is less than the one necessary to produce a nystagmus to the right

We notice periods of bilateral elevation and diminution of the threshold stimulation intensity to the rotatory stimulation On two animals (749, Ge 3) an examination done 2 or 3 days before the reversal of the directional preponderance showed a complete abolition of the nystagmic response, no matter which stimulation intensity we used, no nystagmus could be produced

On the other hand, in cases of spontaneous nystagmus, an antagonistic stimulation must inhibit this spontaneous nystagmus before producing a nystag s ı the other direction The angular acceleration necessary to complete this spontaneous nystagmus corresponds to the acceleration

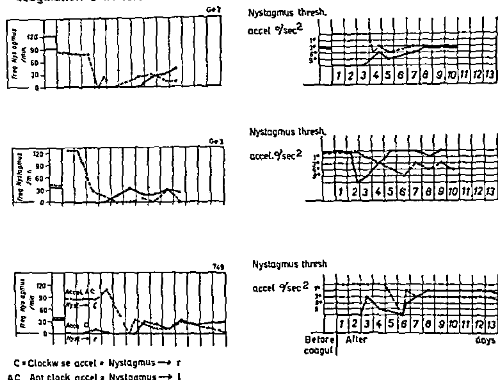


Fig. 4. Action of coagulation of DNA on perrotatory nystagmus. Coagulation of the left DNA of three rabbits (Ge2, Ge3, 749). Daily analysis of nystagmic frequency (clockwise accelerations (C) and anti clockwise (AC) of 3°/s²) and the nystagmic thresholds of liminal rotatory stimulation (intensity of acceleration necessary to produce a nystagmus of regular frequency). On this diagram: Threshold below 1° = spontaneous nystagmus. Threshold above 9° = nystagmus abolished. In the three cases the coagulation produced a directional predominance of the nystagmus towards the side of the lesion (the frequency of the nystagmus beating towards the side of the lesion is higher than that of the nystagmus in the opposite direction). The predominance is accompanied by a parallel variation of the nystagmic thresholds: the threshold of the nystagmus beating towards the side of the lesion is lower than that of the nystagmus of the opposite side. After 4–8 days, tendency towards inversion of the directional predominance.

threshold present before producing the lesion of the diencephalic nystagmogenic area (DNA). If the threshold was 3°/s², after producing a lesion of the DNA, we can mask the spontaneous nystagmus produced with an antagonistic stimulation of 3°/s², while in order to produce a nystagmus in a direction contrary to the spontaneous nystagmus, we need an intensity of 6°/s², 9°/s² or more.

Usually the post lesional electronystagmographic recordings are less homogeneous than those of a normal animal. The frequency and amplitude are frequently irregular. There appear volleys of 5 to 10 beats per second separated by intervals of 4–5 sec. The animal is frequently violently agitated during the stimulation. Furthermore, the distinction between the slow and fast component tends to lessen.

The quantitative analysis of the nystagmographs recorded before and after coagulation of the DNA are presented in Figs 3 They demonstrate the variations of nystagmic frequency produced by clockwise and counterclockwise angular acceleration of 3°/s² as well as the evolution of thresholds to stimulation after coagulation of the DNA

In general the recordings for different degrees of stimulation intensity can be superimposed and the results obtained with a stimulation of 3°/s² are concordant with stimulations of 6°/s² and 9°/s² We notice however some alteration of the rhythm and amplitude depending on the stimulus intensity

In summary unilateral lesions of the DNA are followed by a directional preponderance of the perrotatory horizontal nystagmus towards the side of the lesion This preponderance regresses in 4 to 8 days and is followed usually after the tenth day by a directional preponderance in the other direction that is, to the side contralateral to the DNA lesion

3 *Localization of the Vestibular Nystagmogenic Area (VNA)*

On 12 rabbits we stimulated the region of the superior and medial bulbar vestibular nuclei with the same parameters that were used for the DNA (50/s 0 5 msec 0 7 volt) On 11 of these the DNA was bilaterally stimulated in order to compare the nystagmic responses between the DNA and VNA

(a) *Analysis of responses*

The stimulation frequently produced motor reactions of defense cries rhythmic movements of the jaws and sudden isolated ocular movement usually of ipsiversive character On the other hand in a very well delineated area a selective nystagmic response could be obtained the majority of the points stimulated induced a horizontal nystagmus always ipsiversive (towards the stimulated side) and preceded by a latency period of 1 to 10 sec The nystagmic frequency increased progressively to reach a maximum between the tenth to twentieth sec of stimulation but if the deviation or rotation of the head were well pronounced it sometimes started with a maximum frequency which progressively slowed down whatever the stimulation intensity might be It was usually followed by a nystagmic after discharge in the same direction In addition the free animal presented a contraversive head deviation tendency to contraversive manege and sometimes falling to the side opposite the stimulation

In three rabbits (863 872 868) a vertical nystagmus was obtained The movements of both eyes were perfectly conjugate in the direction of a rotation nd a longitudinal axis With the stimulation of the right side there was a deviation of the right eye downwards and of the left eye upwards and then appeared an upward nystagmus for the right eye and downward for the e The animal rolled in such a way that the ipsilateral ear descended t the ground and it fell to that side

VESTIBULAR NYSTAGMOGENIC AREA

• = Horizontal nystagmus (ipsiversive)

• = Vertical nystagmus

= No response

Fig 5 A

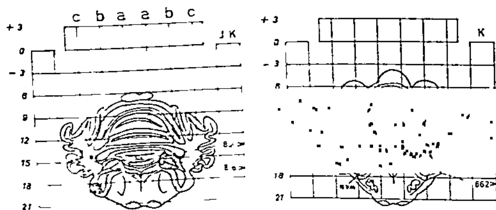


Fig 5 *B*

Fig 5 *A* Stimulated points of the vestibular nystagmogenic area on a parasagittal section of the rabbit's brain (subtentorial extension of the Atlas of Monnier & Gangloff, 1961) ▲ Stimulation points of DNA *B* Lesions following coagulation of optimal nystagmogenic points in the vestibular area ∧ Vertical nystagmus upward >, horizontal nystagmus to the right <, horizontal nystagmus to the left Cb Cerebellum CGM Corpus geniculatum mediale CI Capsula interna CoI Colliculus inferior Fh Fimbria hippocampi NCa, Nucleus caudatus TOp Tractus opticus CGL Corpus geniculatum laterale Cos Colliculus superior CRa Corona radiata GP Globus pallidus, ROp Radiatio optica Flp Fasciculus longitudinalis posterior CR Corpus restiforme (ped cerebell inf) NF Nucleus fastigii NVl Nucleus vestibularis lateralis NVm Nucleus vestibularis medialis NVS Nucleus vestibularis superior Nde Nucleus dentatus NEmb Nucleus emboliformis

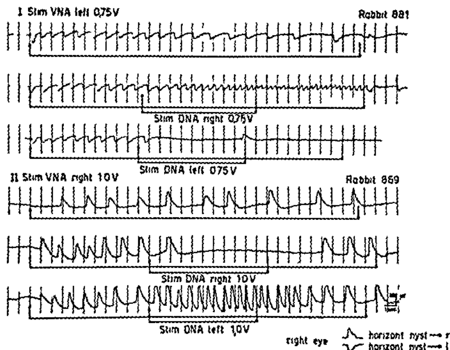


Fig 6 Action of electrical stimulation of the diencephalic nystagmogenic area on the electrical stimulation of the vestibular nystagmogenic area I Stimulation of the left VNA, reinforced by simultaneous stimulation of the right DNA and inhibited by left DNA stimulation II Stimulation of the right VNA inhibited by simultaneous stimulation of the right DNA and reinforced by left DNA stimulation

In one animal (869), a bipolar stimulation by means of two electrodes, one of which had produced a vertical nystagmus and the other a horizontal nystagmus, always resulted in a horizontal nystagmus, no matter what was the intensity or polarity of the stimulus Light had a very strong influence on the nystagmic frequency induced by stimulation of the vestibular nuclei

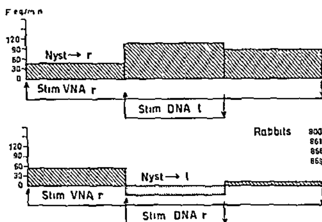
(b) *Stereotaxic localization of the vestibular nystagmogenic area* (VNA) (Fig 5)

The localization of the nystagmogenic points showed a certain concentration 15–16 mm below the inferior surface of the socklet on the sagittal plane ab and the transverse planes JK, K KL, that is in the region of the angle lateral to the fourth ventricle at the level of the brachia pontis

(c) *Anatomic control* (Fig 5)

The optimal nystagmogenic points were coagulated at the end of the experiments in order to observe the results of the lesion on the behavior of the animal and also in order to control their localization The coagulation observed on the histology slides was transcribed on the stereotaxic charts of the rhombencephalic region of Monnier and Gangloff's Atlas In all the animals, the coagulated points were situated in the lateral angle of the fourth

A) Stimulation of the right vestibular nystagmogenic area



B) Stimulation of the left vestibular nystagmogenic area

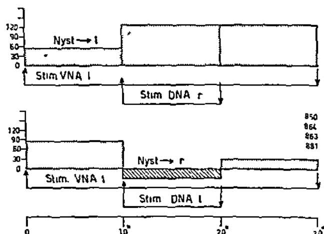


Fig 7 Action of DNA stimulation on VNA stimulation Analysis of the mean *frequency* of nystagmus of eight rabbits before during and after simultaneous stimulation of DNA and VNA

ventricle, at a level corresponding to the medial region of the brachia pontis, they were localized mostly in the superior vestibular nucleus and, to a lesser extent in the medial vestibular nucleus and the cerebellar nuclei

The stereotaxic and anatomic localization of the vertical nystagmogenic points was more lateral and posterior than that for horizontal points

(d) *Results of lesions of the vestibular nystagmogenic area* (*VNA*)

The destruction of the optimal nystagmogenic points produced the following phenomena

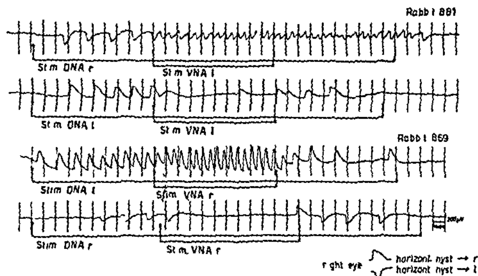


FIG 8 Action of stimulation of the vestibular nystagmogenic area on the stimulation of the diencephalic nystagmogenic area I Stimulation of the right DNA reinforced by simultaneous stimulation of the left VNA and inhibited by stimulation of the right VNA II Stimulation of the left DNA reinforced by stimulation of the right VNA and inhibited by stimulation of the left VNA

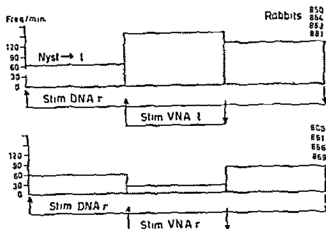
A lesion of the horizontal nystagmogenic points in five rabbits (850, 863, 881, 861, 866) produced either an ipsiversive spontaneous nystagmus or a facilitation of the ipsiversive nystagmus accompanied by a contraversive deviation of the head and the eyes (syndrome deviationnel harmonieux) In one animal (864) only disorganized ocular movements were produced

A lesion of the vertical nystagmogenic points, on the right side (872 868) produced an ipsiversive spontaneous nystagmus (towards the right) combined or alternating with an upward vertical nystagmus for the right eye and downward for the left eye The combination of the vertical and horizontal nystagmus was especially evident during the *rapid* component the eyeball, after an initial horizontal (or vertical) movement suddenly changed direction at a right angle The slow component had an oblique or rotatory excursion

On the other hand, the vertical nystagmus was always associated with a less of head position in the vertical plane

A simultaneous lesion of a vertical and a horizontal nystagmogenic point on the right side of one animal (869) produced an ipsiversive horizontal spontaneous nystagmus (towards the right) associated with a superior spontaneous vertical nystagmus and contralateral manege After 7 days this pattern inverted and the spontaneous nystagmus became horizontal, towards the left

In summary the electrical stimulation of a vestibular nystagmogenic area which included the superior vestibular nucleus of Bechterew and to a lesser degree the medial vestibular nucleus of Schwalbe, produced an ipsiversive horizont[illegible] nystagmus (towards the side of the lesion) On certain occasions, there result[illegible] a vertical nystagmus the eyeball of the stimulated side having



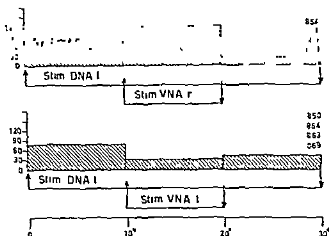


Fig. 9. Action of stimulation of the VNA on stimulation of the DNA. Analysis of the mean frequency of nystagmus on eight rabbits before, during and after simultaneous stimulation of DNA and VNA.

rapid movements upward, the other downward. The coagulation of the VNA tended to produce an ipsiversive or contraversive spontaneous nystagmus, either of a purely horizontal nature, or alternating with or combined with a vertical nystagmus.

4. *Functional and Anatomical Correlations between the VNA and the DNA*

(a) *Histologic study of degenerations secondary to lesions of the DNA*

The optimal nystagmogenic points were coagulated and histological controls showed that they are located in an intermediary and caudal segment of the

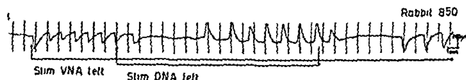


FIG 10 Simultaneous electrical stimulation of DNA and VNA Action of stimulation of the DNA on the nystagmus obtained by stimulation of a horizontal nystagmogenic point of the ipsilateral VNA Inhibition inversion of direction of the nystagmus, recurrence of inhibition preceding the recurrence of the vestibular nystagmus

diencephalon within the intralaminary system, between the lateral geniculate body, the dorsolateral nucleus of the thalamus, the pulvinar, the medial geniculate body and the optic radiation

In five rabbits the secondary degenerations following coagulation of the optimal nystagmogenic substrate were examined with the method of Marchi The coagulation produced a partial destruction of the lateral geniculate body, optic radiation, medial Lamina medullaris, between the lateral geniculate body and the lateral thalamic nucleus, sometimes also part of the neighbouring Cornu ammonis Secondary fiber degenerations could be followed downwards partly towards the tectum (colliculus superior) and partly towards the medial longitudinal bundle and adjacent reticular formations, the medial and superior vestibular nuclei on both sides, the superior cerebellar peduncle on both sides Consequently, direct anatomical connexions seem to exist between the diencephalic nystagmogenic area, the vestibular nuclei, the medial longitudinal bundle and reticular formations on both sides

(b) *Results of simultaneous stimulation of the DNA and VNA*

On eight rabbits, we used the effects produced by simultaneous electrical stimulation of the DNA and VNA

(1) If, during a 30 sec stimulation of the VNA (Figs 6 and 7), we add from the tenth to the twentieth sec a contralateral stimulation of the DNA, the frequency of the vestibular nystagmus is immediately increased by the interaction of the two stimuli, there is no clear cut transition nor any movements of different pattern or rhythm The result is identical to that which would result in a sudden and strong increase of the stimulation voltage As soon as the superimposition is ended, while only the VNA is still stimulated, the nystagmic frequency continues to increase, which corresponds to a persistance of the addition phenomenon during the after discharge period

(2) If, to the VNA stimulation (Figs 6 and 7) we superimpose an ipsilateral DNA stimulation the vestibular nystagmus is abolished At the end of the superimposition, there appears one or more nystagmic beats in the opposite direction usually only after a period corresponding to the persistance of the inhibition effect during the after discharge period does the 'vestibular' nystagmus reappear

(3) If to a 30 sec stimulation of the DNA (Figs 8 and 9), a stimulation

of the contralateral DNA is superimposed between the tenth to the twentieth sec, these stimuli are synergistic and their effects are complementary The nystagmus is greatly increased

(4) If, to a stimulation of the DNA (Figs 8 and 9), an ipsilateral VNA stimulation is superimposed between the tenth to the twentieth sec, the diencephalic nystagmus is inhibited, and reappears only after the end of the interference

We note that, in regard to the horizontal nystagmus, there is an antagonistic function of the homolateral VNA and DNA while the contralateral VNA and DNA are synergistic

On the other hand, if the interfering stimulation is stronger than the primary stimulation, in cases where there would be different threshold stimulation between the two zones (0 5 to 0 7 volt), the simultaneous stimulation of the two antagonistic zones not only masks the nystagmic response but reverses it The nystagmic reversal is preceded by a latency period during which there is no indication of the competition between the two zones (Fig 10) On the contrary, if we stimulate simultaneously a vertical nystagmic point and the DNA (which always produces a horizontal nystagmus), we obtain a mixed nystagmus Instead of the simple phenomenon of addition, there is competition between the horizontal and vertical nystagmus For example, on the animal No 869, during a 30 sec stimulation of the left DNA, the superimposition, between the tenth and twentieth second, of a vertical vestibular nystagmus due to stimulation of the right VNA was realized Instead of a simple addition, there is a competition between the horizontal nystagmus and the vertical the eyeball at first moves half way in the horizontal plane and then suddenly turns upward at a right angle

In summary, the DNA stimulation annuls or reverses the response of the ipsilateral VNA stimulation and strengthens that of the contralateral VNA stimulation If the resulting nystagmus is horizontal, a pure phenomenon of addition or inhibition occurs without completion On the contrary, if one of the stimulated points produces a vertical nystagmus and the other a horizontal nystagmus, there will result a combination of horizontal and vertical nystagmus during simultaneous stimulation

IV. DISCUSSION

1. *Localisation of the Diencephalic Nystagmogenic Area (DNA)*

Our experiences confirmed precisely the delineated localization of the diencephalic nystagmogenic area (DNA) as described by Lachmann, Bergmann & Monnier (1958). With stimulation of low intensity (50 c/s, 5 msec, 0.1 to 1.0 volt) and while eliminating the inhibiting influences of retinal origin by having the animal in complete darkness, we were able to define an area limited to the diencephalon, which corresponds to the zone where *Lachmann, Bergmann & Monnier (1958) had obtained the greatest number* of positive responses. The anatomic substratum of the DNA is located among the structures belonging to the afferent optical pathways (Rob), to its thalamic synapse and the intermediate intralaminar system. We did not find by stimulation a caudal prolongation of this zone into the mesencephalon. However, the study of the descending degeneration does not exclude connections to that region. Let us recall, in regard to this subject, that Monnier (1944) obtained nystagmic responses by stimulation of the mesencephalic reticular substance of the rhesus monkey.

We did not find any nystagmic points in the cerebral cortex nor were we able to demonstrate in the rabbit the presence of a cortical nystagmogenic zone such as that described by di Giorgio (1937) for the Guinea pig. The cortex seems rather to have an inhibitory function upon the DNA. In fact, Bergmann, Lachmann, Monnier & Krupp (1959) demonstrated that section of the afferent cortical fibers produces a facilitation of the DNA stimulation. The nystagmus obtained by electrical stimulation of the diencephalon presents certain constant characteristics: its electric stimulation threshold is very low (around 0.5 volt) in all the rabbits that we examined. It is always horizontal and contraversive, that is, to the side opposite the stimulation. It is always preceded by a latency period and followed by an after discharge in the same direction. It tends to be associated with a cephalic nystagmus or movement of the nictitating membrane, but it is never accompanied by deviations of the trunk or limbs such as those observed by stimulation or lesions of the colliculi and tegmentum (Monnier 1944, 1946, Hess 1954).

2. *Influence of the DNA upon the Perrotatory Nystagmus*

a. The diencephalic nystagmus is in every way comparable to the nystagmus produced by physiological stimulation of the peripheral vestibular apparatus.

The [illegible] rotatory stimulation as described by A. Montandon (1955) using ang[illegible]leration rigorously controlled and measurable produces

a nystagmus with well defined constant characteristics its threshold in the rabbit is between 1° and 3°/s² of angular acceleration The nystagmus beats always in the same direction as the acceleration and the opposite direction of the deceleration We can then postulate that it is always ipsiversive that is towards the side that is stimulated It is preceded by a latency period and followed by a nystagmic after discharge in the same direction Its frequency increases if the *intensity of the stimulation increases*

The identification of the nystagmic responses of the diencephalic and vestibular receptors is not only of a qualitative but also quantitative nature the duration of the latency of the nystagmic after discharge, the frequency and amplitude *are concordant for liminal stimulation as well as for supra* liminal stimulation Let us remark that the frequency of the peripheral nystagmus does not change during the stimulation while that of the diencephalic nystagmus increases then slows down during the stimulation It seems on the *other hand that the animal can withstand better a rapid nystag*mic frequency when it is of central instead of peripheral origin

The characteristics of a post rotatory nystagmus following a sudden stop after a period of constant rotation should not be compared to the nystagmus of *diencephalic origin as to their pattern and their quantitative* evaluation the frequency and amplitude of the post rotatory nystagmus are first of maximum amplitude and gradually diminish in a manner resembling the nystagmic after discharge of a supra liminal stimulation

(b) *The action of DVA stimulation upon the perrotatory and caloric vestibular nystagmus was demonstrated by Lachmann Bergmann Weinmann & Welner (1958)*

The stimulation of the DVA is cancelled by an ipsilateral rotatory or caloric stimulation and strengthened by a contralateral rotatory or caloric stimulation

(c) *Unilateral lesions of the DVA bring about a disorganization of the vestibular system and produce a rotatory directional preponderance towards the side of the lesion*

After coagulation of the DVA the liminal rotatory stimulation demonstrated the presence of an important nystagmic directional preponderance towards the side of the lesion This directional preponderance is compensated in 6 to 8 days after which it tends to reverse itself It is manifest by the presence of a spontaneous or latent nystagmus or by a higher nystagmic *frequency towards the side of the lesion* In the cat Spiegel & Scala (194?) have already observed after unilateral cauterization of the corpus geniculatum laterale and superior colliculus (1946) a very definite directional preponderance of the post rotatory nystagmus towards the side of the lesion They attributed this to the interruption of the inhibitory influence *of retinal* origin upon the vestibular reflex arc It seems most likely then that in doing

their coagulation part of the DNA was injured and that this zone would then be located in the cat in the same region as the rabbit

On the other hand, Carmichel Dix & Hallpike (1954) described an ipsiversive directional preponderance to caloric nystagmus in man with the presence of temporal lobe tumors and conclude the existence of cortical control upon the vestibular reflex arc We cannot exclude the possibility that temporal lobe tumors could act by compression of the immediately underlying DNA

We must note that the nystagmographic recordings *following DNA coagulation* are very deranged Besides bilateral increase of the thresholds we have alterations in the rhythm amplitude and form of the nystagmic movements The whole picture is as if the central mechanism of nystagmus is greatly altered

It seems, however that the *DNA is situated outside the vestibulo ocular* reflex arc of Szentagothai 1950 its presence is not necessary for the demonstration of the nystagmic reflex Bergmann, Lachmann Monnier & Krupp (1959) by successive frontal sections of the mesodiencephalon were able to interrupt the efferent fibers of the DNA without abolishing the postrotatory nystagmus *which was even sometimes facilitated Already* Magnus (1924) had shown that total section of the cerebral trunk at the level of the anterior tubercules quadrijumeaux of the rabbit did not abolish the nystagmus produced by stimulation of the peripheral vestibular apparatus

The fact that lesions of the DNA produce a permanent disruption of the *central mechanism of nystagmus and alter it in a stronger manner that* bilateral exclusion of afferent DNA fibers suggests that the DNA exercises an activator control upon the effector centers for nystagmus of one direction and an inhibitory control for the nystagmus of the opposite direction

3 Localization of the Vestibular Nystagmogenic Area (VNA)

The electric stimulation of the bulbar vestibular nuclei delimited a vestibular nystagmogenic area (VNA)

This area is composed mostly of the superior vestibular nucleus of Bechterew *and in a lesser measure the medial vestibular nucleus of Schwalbe* The parameters of stimulation and the thresholds measured in volts are identical to those of the DNA The nystagmus produced by VNA stimulation is always ipsiversive It has the same characteristics as the diencephalic and per rotatory nystagmus Their quantitative values are identical (duration of *latency and nystagmic after discharge frequency) However while* DNA [illegible] ulation produces a purely nystagmic reaction of the eyes and the head and [illegible] ly in the horizontal plane the VNA stimulation produces a complete syndr[illegible] f [illegible] vational reactions sometimes very violent as well in the vertical [illegible] [illegible] zontal plane The region of the vestibular nuclei belong

in fact to that part of the anatomic substratum producing deviational reactions as Monnier (1944) demonstrated in cat and monkey It is also interesting to note that, in the monkey, Monnier (1944) was able to produce an ipsiversive horizontal nystagmus by stimulation of the posterior rhombencephalic longitudinal bundle, which receives direct fibers from the superior vestibular nucleus

The vertical nystagmus that we obtained in some cases presented exactly the same characteristics as the horizontal nystagmus, conjugate movements of both eyes of a rotatory nature around a longitudinal axis, the eye of the stimulated side beating upward, the other, downward The points for the vertical nystagmus are more lateral than the spontaneous vertical nystagmus in the region of the restiform body If the head is not fixed, it rotates in the same direction as the slow component of the nystagmus The vertical nystagmus with an eye beating superiorly and the other inferiorly, is very characteristic of the rabbit, it is also found after labyrinthectomy (Aubry & Pialoux, 1957)

Vestibular nystagmogenic area coagulation produces spontaneous ipsiversive nystagmus, that is, in the same direction as that obtained by electrical stimulation It is possible that the lesion creates an irritative state having stimulating effects upon the efferent pathways or, on the contrary, suppresses some important cerebro vestibular inhibitory pathways

Let us recall again that spontaneous nystagmus is of a mixed nature, with vertical and horizontal movements, often very disorganized, irregular and intermittent This seems due to the irritative rather than the destructive properties of the lesion This is in accord with the observations of many researchers In fact, while the labyrinthectomy brings about a unilateral ipsiversive deviational syndrome and a contraversive spontaneous nystagmus (syndrome vestibulaire harmonieux) the lesions of the vestibular nuclei produce different reactions according to their localization In the guinea pig, Buchanan (1940) selectively destroyed the lateral vestibular nucleus (LVN) of Deiters, the medial vestibular nucleus (MVN) of Schwalbe and the nucleus of Roller The LVN coagulation is followed by a syndrome vestibulaire harmonieux identical to labyrinthectomy of the same side, that is, an ipsiversive or vertical deviation of the eyes and the head with a contraversive or vertical nystagmus The MVN coagulation produces a syndrome in the opposite direction contraversive deviation and ipsiversive nystagmus The destruction of Roller s nucleus gives an inconstant picture

In the cat Monnier (1943) coagulated the SVN of Bechterew the pedunculo protuberantial isthmus and the fasciculus longitudinalis posterior All of these lesions were followed by a contraversive deviational syndrome The results obtained by our coagulations are in agreement with those obtained by Monnier and Buchanan after lesions of the MVN and SVN, but differ from those of the LVN It then seems most likely that the VNA is limited to the SVN and MVN and does not include the lateral vestibular nucleus of Deiters

4 Functional and Anatomic Correlations between the DNA and VNA

(a) *The histologic study of myelin degeneration following DNA* allows us to postulate the possibility of direct anatomic connections between the DNA and VNA of each side by means of the posterior longitudinal tract (l lp)

(b) *The DNA inhibits the ipsilateral VNA and enhances the contralateral VNA*

Our experiences with interference show the DNA stimulation annuls or reverses the nystagmogenic effects of the VNA if both zones are simultaneously stimulated on the same side and on the contrary, strengthens them if the simultaneous stimulations are crossed This interference intervenes without any disturbances the nystagmus slows down and then stops, as if the stimulation intensity would be diminishing, and then it reverses itself, or, on the other hand, it rapidly increases as if the stimulus intensity were doubled or trebled There is no modification in the pattern of the nystagmic movements nor any hesitation period or combination of many different movements The two stimulations act most likely by the same effector system On the contrary the simultaneous stimulation of vertical and horizontal nystagmogenic points produces mixed responses, with nystagmic movements composed of many movements in different directions as if there were competition between two different effector mechanisms

Only nystagmic reactions of the eyes and the head are modified by the DNA during interference experiments

The experiments of simultaneous DNA and VNA stimulations gave results identical to those obtained by Lachmann, Bergmann, Weinmann & Welner (1958) adding DNA stimulations to the perrotatory and caloric nystagmus

Many indices seem to suppose the existence of a single effector system for the horizontal nystagmus

— The nystagmic responses to rotatory stimulation of the peripheral receptor and the electrical stimulation of both the DNA and VNA are identical

— The simultaneous stimulation of the peripheral receptor and the DNA or of the VNA and DNA interfere by simple addition or masking of the nystagmic effects, without any competition phenomena

Bergmann Lachmann Monnier & Krupp (1959) proposed the hypothesis that the diencephalic and vestibular nystagmic influxes meet in a common final substratum situated in an area including the oculo motor nuclei and the nuclei of Darkschewitsch and Cajal

Already Muskens (1934) had said that the nystagmus was integrated and organized in the *nuclei of the* posterior commissure and Lorente de No (1938) had insisted on the importance of the reticular formation in the mechanism of nystagmus On the other hand, it seems most improbable that the effector center of nystagmus is situated in the DNA itself since its exclus[illegible] transverse section of the cerebral trunk does not abolish the perrotat[illegible]gmus Magnus (1924) Bergmann Lachmann Monnier &

Krupp (1959)] The superior and medial vestibular nuclei also do not appear to represent the total of the effector system, since their coagulation, instead of abolishing the nystagmus, produces a spontaneous nystagmus of the same direction as that produced by an irritative stimulation We can then admit that the mechanism of nystagmus is accomplished by a complex system between the common oculomotor nuclei and vestibular nuclei, with the DNA having an activating or inhibiting function upon this system

V SUMMARY AND CONCLUSIONS

Using the technic and Stereotaxic Atlas of *Monnier & Gangloff* (1961) we have confirmed and delineated the localization of the diencephalic nystagmogenic area (DNA) in the rabbit, as described by Lachmann, Bergmann & Monnier in 1958.

The electrical stimulation of the caudal, dorsal and lateral region of the diencephalon, in the neighborhood of the lateral geniculate body and the optic radiations, with a frequency of 50 impulses per sec, a duration of 0.5 msec and an intensity of 0.1 to 1 volt, produces a contraversive horizontal nystagmus. This diencephalic nystagmus has the same appearance and the same characteristics as the vestibular nystagmus of peripheral origin.

Unilateral destruction of the DNA modifies the nystagmic response of the peripheral vestibular apparatus, which we controlled by the liminal rotatory stimulation described by A. Montandon (1955). In the days immediately following the lesion there is an important directional preponderance towards the side of the lesion; this is compensated at the end of one week and then tends to reverse itself. The post lesional nystagmus also presents alterations of pattern, frequency and amplitude.

The electrical stimulation in the region of the vestibular nuclei, with the same method and parameters as for the diencephalon, produces a horizontal nystagmus with a rapid ipsiversive component and, in some cases, a vertical nystagmus. The vestibular nystagmogenic area (VNA) is located in the lateral angle of the fourth ventricle and includes the superior and medial vestibular nuclei.

We have studied the anatomic connections between the DNA and VNA by the Marchi method. To analyze the functional connections between these two centers, we stimulated simultaneously both of them with the same parameters. When the two stimulated centers were on the same side, their nystagmic effect was cancelled, while on the contrary, the action of one was added to the action of the other if they were on different sides.

We conclude from our experiments:

— that the electrical stimulations of the DNA and contralateral VNA produce the same nystagmic response as the physiologic stimulation of the contralateral peripheral receptors.

…at stimulation of the DNA attenuates the response of the ipsilateral …lar nuclei and strengthens that of the contralateral vestibular nu…

— that the destruction of the DNA produces a disorganization of the nystagmic response and is followed by a directional preponderance of the nystagmus

The existence of bilateral connections between the DNA and VNA was confirmed by the histologic study of myelin degeneration by the Marchi method

ZUSAMMENFASSUNG

Unter Verwendung der Technik und des Atlasses von Monnier & Gangloff (1961) haben wir die Lokalisation der von Lachmann, Bergmann & Monnier (1958) beschriebenen nystagmogenen diencephalen Area (DNA) beim Kaninchen bestätigt und präzisiert.

Die elektrische Reizung dieser Area bewirkt einen horizontalen entgegengerichteten Nystagmus vergleichbar dem durch die physiologische Reizung des kontralateralen horizontalen Bogenganges hervorgerufenen Nystagmus.

Die einseitige Zerstörung dieser Area modifiziert den Nystagmus-Effekt des peripheren vestibulären Apparates, getestet mittels der „Epreuve giratoire liminaire" von A. Montandon. Es kommt zu einer Verstärkung des Nystagmus nach der Seite der Läsion, welche sich nach einiger Zeit kompensiert und umkehrt.

Die elektrische Reizung des Vestibulariskerngebietes bewirkt einen horizontalen Nystagmus zur stimulierten Seite gerichtet, und in einigen Fällen einen vertikalen Nystagmus. Die nystagmogene vestibuläre Area (VNA) enthält den Nucleus vestibularis superior und den Medianus.

Wir haben die anatomischen Verbindungen der beiden nystagmogenen Areen nach der histologischen Methode von Marchi untersucht, dann die beiden Areen zwecks Analyse ihrer funktionellen Verbindungen simultan mit den gleichen Parametern elektrisch gereizt.

Wenn die beiden stimulierten Areen gleichseitig lokalisiert sind, heben sich ihre nystagmogenen Wirkungen auf. Im Gegensatz dazu addieren sie sich, wenn die Reizungen gekreuzt durchgeführt werden.

Die diencephale Area dämpft die nystagmogene Wirkung der ipsilateralen Vestibularis-Kerne und verstärkt diejenige der kontralateralen.

REFERENCES

AUBRY and PIALOUX Maladies de l oreille interne et oto-neurologie Masson, 1957

BERGMANN, F, LACHMANN, J, MONNIER, M, and KRUPP, P Central nystagmus III Functional correlations of mesodiencephalic nystagmogenic center *Am J Physiol*, *197*, 328, 1958

BUCHANAN, A R Nystagmus and eye deviations on guinea pigs with lesions in the brain stem *The Laryngoscope 50*, 1002, 1940

CARMICHAEL, E A, DIX, M R, and HALLPIKE, C S Lesions of the cerebral hemispheres and their effects upon optokinetic and caloric nystagmus *Brain*, *77*, 345, 1954

DI GIORGIO, A M *L'Apparell vestibulaire* Presses Univ de France, 1957

FRANCESCHETTI, A, MONNIER, M, MONTANDON, A, and DIETERLE, P Standardisation des inscriptions nystagmographiques, II[e] Symposium Vestibulaire International, Genève, 1959 *Confin neurol*, *20*, 275–276, 1960

HESS, W R *Die Methodik der lokalisierten Reizung und Ausschaltung subkortikaler Hirnabschnitte* Thieme, Leipzig 119, 1932

HESS, W R *Diencephalon* Grune, New York, 79, 1954

JUNG, R Eine elektrische Methode zur mehrfachen Registrierung von Augenbewegung und Nystagmus *Klin Wschr*, *1*, 21–24, 1939

JUNG, R, and TOENNIES, J F *Klin Wschr*, *26*, 513, 1948

LACHMANN, J, BERGMANN, F, and MONNIER, M Central nystagmus elicited by stimulation of

LEKSELL L Clinical recording of eye movements *Acta Chir Scand*, Stockholm, *82*, 262–270, 1939

LORENTE DE NO, R *J of Neurophysiol*, *1* 207 1938

MAGNUS R *Korperstellung* Springer, Berlin, 1924

MEYERS, I L Nystagmography *Arch Neurol 21* 901 918 1929

MONNIER, M Syndromes deviationnels provoques par excitation et destruction du système reticulaire bulbo protubérantiel chez le chat (Manege et reaction tegmentale) *Mschr Psychiat Neurol*, *107*, 84–102, 1943

MONNIER M Physiologie du tronc cérébral (Le role du système réticulaire dans l organisation de la motricité extrapyramidale) *Ergeb Physiol 45* 321–422 1944

MONNIER, M Les formations reticulaires tegmentales Equilibration des postures du regard, de la tête et du tronc *Rev Neurol 78* 422–452 1946

MONNIER, M and HUFSCHMIDT H J Das Elektro Okulogramm (EOG) und Elektro-Nystagmogramm (ENG) beim Menschen *Helv Physiol Acta*, *9* 348 1951

MONNIER, M and LAUE H Technique d enregistrement electrique du nystagmus vestibulaire rotatoire chez l animal *Helv Physiol Acta 10* C20–C22 1952

MONNIER M and GANGLOFF H *Atlas for Stereotaxic Research on the Conscious Rabbit* Elsevier, 1961

MONTANDON A and MONNIER M Analyse du nystagmus vestibulaire par la methode électronystagmographique *Ann Oto-Laryng 68* 701 765 1951

MONTANDON A MONNIER M CROCI and BRUNNER W Enregistrement simultané des variations de la pression arterielle et du nystagmus provoqués par la stimulation vestibulaire giratoire *Confin neurol 12* 255 1952

MONTANDON A A new technique for vestibular investigation Proc. collegium oto-rhino-laryng London 1954 *Acta otolaryng* Stockholm, *44*, 594 596 1954

MONTANDON, A , and DITTRICH, F Importance d un contrôle accéléro métrique et tachymétrique permanent et direct du plateau en nystagmographie perrotatoire *Acta oto-laryng belg*, *15*, 150–157, 1961

MOWRER, O H , RUCH, T C , and MILLER, N E *Am J Physiol* , *114*, 423, 1936

MUSKENS, L J J *Das Supra Vestibulare System* N V Noord Hollandsche Uitgevermaatschappij, 1934

SPIEGEL, E A and SCALA, N P Effects of lesions of the optic tract on the internal geniculate body *Confin Neurol* , *7*, 68–76, 1946

SPIEGEL, E A , and SCALA, N P Effects of quadrigeminal lesions upon labyrinthine nystagmus *Confin Neurol* , *7*, 68–76, 1946

SZENTAGOTHAI, J *J Neurophysiol* , *13*, 395, 1950

WYSS, O A M Lin Hochfrequenz Koagulationsgerat zur reizlosen Ausschaltung *Helv Physiol Acta*, *3* 437–443, 1945

*

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1964

Acta

OTO-LARYNGOLOGICA

S U P P L E M E N T U M 187

THE INNER EAR IN GENETICALLY DETERMINED DEAFNESS

Report and Analysis of 2 New Cases

BY

FRANZ ALTMANN

ACTA OTO-LARYNGOLOGICA · KARLAVÄGEN 41, STOCKHOLM Ö

From the Department of Otolaryngology, Columbia University, College of Physicians & Surgeons

ACTA OTO-LARYNGOLOGICA

SUPPLEMENTUM 187

THE INNER EAR IN GENETICALLY DETERMINED DEAFNESS

Report and Analysis of 2 New Cases

BY

FRANZ ALTMANN

M.D., NEW YORK CITY

CLINICAL PROFESSOR OF OTO LARYNGOLOGY

COLUMBIA UNIV. COLLEGE OF P. & S.

UPPSALA 1964

This work was supported by Grant B 3376
of the United States Public Health Service

PRINTED IN SWEDEN BY
Almqvist & Wiksells
BOKTRYCKERI AKTIEBOLAG
UPPSALA 1964

Dedicated to

Professor Dr. Ludwig Horbst

of Innsbruck, Austria, in honor of his 60th birthday

The anatomical findings in what is commonly called congenital deafness (i e deafness present at birth) can be divided into two groups One group with regressive changes in the epithelial structures of the cochlear duct the saccule and occasionally the utricle their nerves ganglia and central pathways and another group with evident anatomical anomalies of the cochlea and sometimes also of the other parts of the inner ear

The regressive changes characterizing the first group develop either spontaneously without any apparent extrinsic cause or secondary to known extrinsic factors such as virus infections or hemorrhages Spontaneous regressive changes and outright developmental anomalies form the basis of genetically determined (inherited) deafness secondary changes the basis of acquired deafness

The majority of the more severe malformations of the inner ear in genetically determined deafness is found without changes in other parts of the ear Some of the inner ear malformations occur with the group of combined malformations of the external and middle ear known as atresia auris congenita still others are observed in nonviable fetuses associated with developmental anomalies of the central nervous system alone or combined with additional malformations of the external and middle ear

A clearcut distinction between primary (spontaneous) and secondary (regressive) changes as pointed out in a previous paper (Altmann 1950) is often much easier in theory than in practice especially when the findings are limited to the epithelial structures of the cochlea and of the saccule

Lately the interpretation of the anatomical findings in these cases has been facilitated by the investigations of Lindsay and his school on virus diseases of the inner ear and particularly by the rapidly increasing knowledge of the morphology of inherited deafness in mice and other rodents

The findings in these are in many respects very similar or even identical with those in Man, cats dogs or other animals (see Altmann 1950 1951, Wilson & Kane 1959 Hudson & Ruben, 1962) except for the fact that in the majority of the strains of deaf rodents the hearing loss is associated with locomotor anomalies of a kind so far never observed in Man, cats or dogs It is still not clear whether these locomotor anomalies are due to changes in the labyrinth in the central nervous system or in both

Nevertheless the study of the inner ear changes in deaf rodents has given us a much better understanding not only of their specific morphology but also of the functional interdependence of the various epithelial structures of the cochlear duct

So far the observations in spite of their obvious great significance for a better understanding and interpretation of the findings in inherited deafness in Man have found attention mainly from geneticists and physiologists but not from otologists. Very seldom in the reports of human cases is reference made to the findings in rodents.

Under these circumstances a more detailed review of the findings in mice seems justified.

In analogy to the findings in Man *two main groups* can again be distinguished.

Group one comprises animals with morphologically normal inner ears at birth. Degenerative changes develop after birth particularly in the stria vascularis, the organ of Corti and the spiral ganglion (scala media complex, Grüneberg). In some strains the sensory epithelia of the maculae and cristae become also affected.

So far 10 mutants belonging to this group have been examined histologically (see Deol 1956, Kocher 1960). According to the behavior of the animals three subgroups can be distinguished.

In *subgroup one* (shaker 1, shaker 2, jerker, waltzer, pirouette, varitint waddler) the hearing loss becomes manifest at or soon after birth and all the animals show locomotor anomalies.

In *subgroup two* (deaf, deafness) the deafness again becomes manifest soon after birth but locomotor anomalies are absent.

In *subgroup three* (B wh) the hearing loss does not become manifest until after puberty, locomotor anomalies are again absent.

The causal relationships between the changes in the various structures of the inner ear are, as Grüneberg points out, not yet clear. The lesions of the scala media complex always appear together and thus obviously form some kind of causal unit; the degeneration of the spiral ganglion is presumably a consequence of the degeneration of Corti's organ. How these lesions link up with the changes in the stria vascularis is not clear. The hypothesis put forward by earlier authors, that the degeneration of Corti's organ may be secondary to the changes in the stria vascularis—the source of endolymph—is no longer tenable, as similar degenerations are now known to occur in the maculae and other structures that have a blood supply of their own and thus presumably do not depend for their nutrition on the endolymph. However, the fact appears worthwhile mentioning that the endolymph contained in the saccule, the utricle and the membranous semicircular canals is produced in certain parts of the sensory endorgans of these structures and that in the cases with collapse of the saccule and of the utricle the endorgans always show marked degenerative changes. It seems, therefore, that the assumption of earlier authors, notably of van Lennep, could, although with modifications, still be valid. Almost all these mutants show a recessive hereditary trait.

In Man the [illegible] picture of the majority of the reported observations of inherited deafness is either practically identical with that found in deaf

rodents or shows a combination of the degenerative atrophic changes with changes which were called posthydropic neuroepithelial degeneration by Wittmaack they consist in ectasia or collapse of Reissner s membrane a turning over separation or adherence to surrounding formations of the lamina tectoria formation of a nuclei containing sheath around the lamina tectoria complete disappearance of Corti s organ or its transformation into a low mound consisting of flat or more cuboidal cells development of formations resembling epithelial proliferations in the stria vascularis or the development of thin strings between different parts of the cochlear duct Changes might also be found in the maculae of the saccule and the utricle but not as extensive as those in the cochlea

The just described changes were regarded as characteristic for a healed serous labyrinthitis secondary to an infection of the middle ear or of the meninges Lately they have also been found in virus diseases of the inner ear (maternal rubella Lindsay *et al* measles Lindsay & Hemenway mumps Lindsay Davey & Ward)

The histologic differentiation between spontaneous and secondary regressive changes is very difficult if not impossible in many instances particularly in cases in which the latter are superimposed on developmental anomalies This becomes for instance apparent in a case of cochlear malformation of Mondini s type in a newborn child with congenital atresia of the external auditory meatus and internal hydrocephalus reported by the author (1933) in which typical posthydropic neuroepithelial changes were found in the cochlear duct

It seems significant that in all the 10 mice mutants comprising the first main group the inner ear was normal at birth and that the degenerative atrophic changes had started as a rule at birth or soon thereafter but sometimes even much later

On the other hand the term congenital deafness frequently used for the human cases implies that the changes were believed to have started in utero Although this might have been so in some of the observations it is well conceivable that in others in analogy to the findings in mice the changes had set in at or after birth The difficulties in detecting and accurately defining a hearing loss in very young infants together with the small number of histologically examined ears make it at the present time impossible to decide whether there is any essential difference in the time of onset *of the regressive* changes in rodents and in Man

In Man one usually distinguishes between cases with epithelial changes in the cochlear and the vestibular portion of the labyrinth (Siebenmann Bing type) and epithelial changes confined to the cochlear duct and saccule (Scheibe s type) Only two cases clearly belonging to this group and both representing Scheibe s type have come to the author s attention since 1950 The observation of Fisch (1955) concerns a child with deafness associated with Waardenburg s syndrome The inner ear changes were purely degenerative atrophic in nature the brain was found without pathology Buch &

Jorgensen (1963) *report the findings in a deaf mute pregnant woman of 37* years who died after a Caesarean section six weeks before the expected term with delivery of a baby with myelomeningocele, a large hydrocephalus and *Arnold Chiari's syndrome*.

The histological examination of the cochlea of the mother showed a partial collapse of the cochlear duct and a hardly recognizable organ of Corti. The epithelial lining of the stria vascularis was markedly proliferated, in several sites detached from the underlying tissues of the ligamentum spirale and in several places adherent to the "remnants" of the organ of Corti and Reissner's membrane. The stria was very poorly vascularized but where capillaries were present they were normal. The tectorial membrane was present only as remnants, in several places rolled up and usually *in contact with the epi*thelial bridges of the proliferated *stria vascularis*. Spherical periodic acid Schiff (PAS) positive *globules* were present—mainly in an intraepithelial *situation—in the stria, in the partially collapsed saccule and the endolympha*tic duct.

The positive PAS reaction indicates according to the authors the presence *of polysaccharides*, mucopolysaccharides, mucoproteins, glycoproteins, glycolipids, unsaturated lipids or phospholipids. However, in the celloidin sections used by the authors all the lipids must already have been extracted during the process of embedding.

The number of nerve fibers in the lamina spiralis was reduced, the number of ganglionic cells in the modiolus somewhat reduced and there was a fairly ample interstitial fibrosis in the cochlear portion of the acoustic nerve. The brain showed congestive and anoxic changes only.

The same PAS *positive spherical* structures *were also found* by the authors on reexamination of the case of deaf mutism with retinitis pigmentosa reported by Nager in 1927.

It might be pointed out that similar spherical structures in the stria vascularis of deafmutes were already described and interpreted as calcifications by Wolff (1949), by the author in two cases (Altmann 1950) and by others and interpreted either as hyaline or calcified thrombi in the strial vessels or as the result of degenerative changes in the capillary endothelia. Typical *calcium concrements in the stria were observed without any other labyrin*thine changes by Rollin (1934) and by Yamakawa (1938) in a case of true Menière's disease with labyrinthine hydrops. "Phleboliths" were seen by Wolff (1949) in the capillaries beneath the crista of the superior vertical canal in a case with fracture of the skull 5 days prior to death. *However*, in none of these cases a PAS reaction had been performed.

Although the findings *described* by Buch and Jorgensen must not necessarily represent inherited changes, such an assumption is made probable by *the fact that the patient had given birth to a child with multiple malforma*tions.

Ward, Kinney & Lindsay (1962) report the findings *in* the inner ear and the brain of a deaf boy of 17 years; they again were confined to the cochlea

saccular portion There was collapse of Reissner s membrane partial collapse of the saccule marked spotty aplasia or atrophy of the stria vascularis throughout the cochlea almost complete absence of the organ of Corti the tectorial membrane was either compressed into the remaining cellular elements of the organ of Corti or it was rolled up in contact with the limbus either in the sulcus or it rested on top of the limbus It was covered by a single layer of flattened cells The peripheral cochlear neurons and the ganglionic cells of the basal and lower middle coil on one side and of the basal coil only on the other side were diminished in numbers and showed asymmetry of distribution Examination of the brain revealed no abnormalities Since the tectorial membrane was rolled up against the limbus and was covered by a layer of flat epithelial cells a finding encountered in many types of experimental animals and in Man after viral labyrinthitis it is in the opinion of Lindsay more probable that this case represents an acquired rather than an inherited type of deafness

The comparison of this case with the one reported by Buch and Jorgensen shows clearly the difficulties encountered in the interpretation of the findings and the differences in the opinion of different authors

A case belonging to this group in which the interpretation of the findings was less difficult was recently examined by the author through the courtesy of Dr Martin Spector of Philadelphia Dr Spector had observed the patient clinically for several years had procured the temporal bones and sent them to the Department of Otolaryngology College of Physicians & Surgeons of Columbia University for histologic examination

CASE REPORT

Case 1

A N a 7½ year old Puerto Rican girl known to be deaf since the age of 3 years who died in St Christopher s Hospital for children in Philadelphia of staphylococcic septicemia following an operation for patent ductus arteriosus Parents hear well the family history regarding deafness is negative The delivery was normal and so was the development of the girl except that she only spoke a few words like mom and pop The child had never been seriously ill The clinical ENT findings were essentially negative Audiograms showed a severe neurosensory hearing loss on both sides (loss for a c at 2 0 d v 80 db at 500 d v 90 db and at 1000 d v 100 db)

Histologic examination of the temporal bones (fixation 10 % Formalin embedded in Celloidin cut in *20 μ sections* stained with Hematoxylin Eosin)

The *right temporal bone* was cut horizontally the *left temporal bone* vertically the findings were identical on both sides



Fig. 3. Case 1. Right side. Lower middle turn. Absence of Corti's organ. Remnant of tectorial membrane (*m*) turned downward toward the basilar membrane.

extends from a small, on cross section triangular, hillock overlying the lateral portion of the bony spiral lamina in a supero-lateral direction towards the uppermost portion of the spiral ligament (Fig. 4). The medial part of the crista spiralis rests on the lateral slope of the hillock and is in this way elevated and separated from the bony spiral lamina through the tissue of the hillock. The spiral ligament shows at the place of insertion of the connective tissue strand an elevation which on cross section also has a triangular shape.



Fig. 4. Case 1. Left side. Lowermost part of basal cochlear turn. Strand of connective tissue traversing the lumen of the scala vestibuli.



Fig 5 Case 1 Right side Outer wall of the collapsed saccule (*w*) overlying the macula Concrements within the macula (*c*)

The *saccule* is collapsed, the epithelium of the macula is uniformly cubiodeal and no distinction can be made between hair cells and sustentacular cells, the otolithic membrane is missing The epithelium contains in some places the same homogenous pink staining globules, as the stria vascularis (Fig 5) The number of the saccular nerve fibers is normal

The *utricle*, the *membranous semicircular canals*, the *endolymphatic duct* and the adjacent part of the *sac* contained in the specimen fail to show any *abnormalities*

COMMENT

In the just described case degenerative atrophic changes were present in the stria vascularis, the organ of Corti, the spiral ganglion of the lower basal cochlear turn, and in the macula sacculi, with collapse of Reissner s membrane and of the saccule There were nowhere any changes which could be interpreted as developmental anomalies, with exception of the strand of tissue in the lowermost portion of the basal cochlear turn, or as evidence of a healed serous or viral labyrinthitis The findings very closely resemble those in Group I, Subgroup II of the mice where the hearing loss becomes manifest soon after birth but where locomotor anomalies are absent

Under these *circumstances* the diagnosis of *inherited recessive deafness*

(sporadic deaf mutism) of the *cochleo saccular type* (Scheibe) can be made with a reasonable degree of certainty

The cochlear findings obviously represent secondary changes in a normally developed cochlear duct No clue can be gained from the findings regarding the evidently genetically determined time of onset of the degenerative changes nor regarding the relationship of the changes in the various components of the scala media complex to each other The same is true of the spherical PAS positive *globules Their nature* was a little but better but by *no means* satisfactorily defined by their staining reaction with Toluidin blue and methylene blue of pH_4

The strand of tissue traversing the lowermost portion of the basal cochlear turn is evidently a remnant of the embryonic connective tissue which originally filled both cochlear scalae No similar case could be found in the literature but Alexander (1924) mentions that occasionally a strand of connective tissue is seen extending in the apical turn from the floor of the scala tympani to the free edge of the hamulus He explains the condition as an *embryological residuum due to incomplete resorption of the mesenchyme* during the formation of the lumen of the scala tympani

Group two of the mice comprises animals with morphological defects in the inner ear which arise in early stages of embryonic development and which are associated with locomotor anomalies In contra distinction to group one only insignificant or no degenerative changes at all occur in the cells of the sense organs once they have been formed

Seven mutants, again with a recessive hereditary trait, have so far been histologically examined (*Shaker short Kreisler Dreher, Fidget Twirler Zigzag Pallid*) Only the first three strains presenting severe degrees of malformations are deaf the other four presenting lesser degrees of malformations show normal hearing

In *shaker shorts* (Bonnevie 1936) the inner ear consists of a simple vesicle without endolymphatic duct and sac, without semi circular canals and without division into saccule and utricle The ventral part of the vesicle shows however a trace of a spiral curving reminiscent of *a cochlea with a rudimentary organ of Corti on its inner side*

The underlying disturbance is according to Bonnevie, an anomaly of the myelencephalon with an increase in the intracranial pressure which leads on the eighteenth day of embryonic development to a rupture of the roof of the mesencephalon and of the cerebellum with formation of brain hernias

The brain anomaly first becomes manifest during the ninth day of embryonic development and up to that time the development of the ear has progressed to the *formation of normal otocysts From that day on* however, the shape differentiation of the ear vesicle takes an abnormal course, evidently because of pressure changes in the surrounding tissues which lead to an abnormal position of the otocysts

Nevertheless the differentiation of the epithelium of the otocyst into

cristae, maculae and the organ of Corti takes place in a normal way, although at a somewhat slower rate than in normal vesicles The rupture of the roof of the brain breaks or at least seriously disturbs the acoustic nerve centers From then on, possibly owing to the lack of nerve supply, a progressive degeneration of the nerve endings sets in

In *kreislers* (Hertwig, 1944) the sacculus and utriculus are only incompletely separated from each other, the endolymphatic duct and sac are absent, the semicircular canals often defective The cochlear duct forms a wide, slightly curved sac The maculae and cristae are not recognizable, the organ of Corti and the spiral ganglion are absent The cochlear duct is herniated through the internal auditory meatus into the subarachnoid space and cysts are formed in the cerebellopontine angle region which may attain considerable size and may interfere with the development of the adjacent parts of the brain

The normal differentiation of the otic vesicle is dependent on the presence and intactness of the medulla oblongata The underlying disturbance in kreislers is according to Hertwig an invagination of the otic pit at a greater distance from the neural tube than in normal embryos

In *Drehers* (Fischer, 1956) two types of changes can be distinguished

In the more common *type I* utricle and saccule form a wide undivided sac The ampullae of the two very wide vertical canals, and as a rule the entire lateral semicircular canal and the proximal part of the common crus of the vertical canals are incorporated into this sac The endolymphatic duct originates from the saccular portion of the sac and is wide and short The endolymphatic sac reduced in size and lined with flat epithelium The maculae are displaced and the sensularia absent The hair and supporting cells are well preserved The cristae ampullares are flattened, the ampullae reduced in size, the epithelium of the plana semilunata flattened

The cochlear duct has 1 and ¼ instead of 1 and ½ turns, is very wide, particularly in its upper turn and in wide communication with the utriculo saccular sac The organ of Corti is distended but the hair cells are quite well preserved The stria vascularis is degenerated or absent The cochlear duct has been herniated through the basilar membrane and through the fundus into the internal auditory meatus and forms a cyst which displaces the fibers of the cochlear nerve, but only rarely compresses the brain stem

In the less frequent *type II* utricle and saccule again form a common sac which is in wide communication with the cochlear duct The maculae are normal but the otolithic membranes absent The membranous semicircular canals are narrow, the cristae and the cupulae normal The endolymphatic duct is dilated and originates from the saccular portion of the common sac, the endolymphatic sac is lined with flat epithelium The structure of the cochlea is normal, but Reissner s membrane has collapsed, the stria vascularis is hypoplastic or atrophic, the organ of Corti degenerated Reissner s membrane of the basal cochlear turn has been invaginated through the wide cochleosaccular opening into the utriculo saccular sac and often compresses

the maculae. Evidently the malformed endolymphatic labyrinth has become secondarily compressed by a surplus of perilymph.

It must at present remain undecided whether the anomalies in the Drehers are caused in a similar way as in the kreislers by an invagination of the otic pit too far away from the neural tube with subsequent disturbances of the inductive influences emanating from the chordamesoderm or from the neural tube or if they are connected with brain anomalies (hydrocephalus) which are present to a varying extent in all the animals (Bierwolf).

Another recessive mutant, *Shaker with syndactylism* (Hertwig 1951), although actually not belonging to this group, shows great similarities with the type II of the Drehers. The labyrinth is more or less normal at birth. From about the third day onward there is a progressive atrophy of the membranous labyrinth which leads to narrowing of the semicircular canals, of the sacculus and utriculus and of the cochlear duct. Reissner's membrane collapses and is in contact with the tectorial membrane and a narrow lumen remains only in the neighborhood of the stria vascularis. Later that structure and Corti's organ also degenerate.

The next four mutants show normal hearing.

In *Fidgets* (Truslove 1956) the anomalies are confined to the semicircular canals and the immediately adjacent regions. The lateral canal with its crista ampullaris is always absent, the vertical canals are rudimentary or absent but their cristae ampullares show a normal structure and are situated in a fairly normal position except that they are not located in separate ampullae but within the utricle. The endolymphatic duct and sac as well as the pars inferior of the labyrinth are normal.

In *Twirlers* (Lyons 1958) saccule, utricle, endolymphatic duct and sac show normal shape. The lateral semicircular canals are often shortened and the cristae abnormal in shape, forming a pit rather than a ridge. The finer structure including that of the cupula is, however, normal. The lumen of the membranous vertical canals varies in diameter and sometimes diverticule like projections are present which either consist of connective tissue only or contain an actual outpouching of the canal lumen. The cristae ampullares are normal. The cochlea is as a rule normal, occasionally there is a hydrops of the entire cochlear duct or of its apical portion with absence of the stria vascularis and disorganization of the organ of Corti in hydropic areas.

In *Zig-zags* (Lyon 1960) there is either a constriction or a defect of the horizontal canal on one or both sides. There were all grades of defects from complete absence of the canal to normality. Intermediate grades do not consist of shortening of the canal but of a constriction in the middle of its length or of a defect of the middle portion leaving blind ending stumps at both ends. The ampulla of the canal is always present and the crista ampullaris normal. The other parts of the labyrinth are completely normal.

In *Pallid* (homozygotes for the mutant pallid which dilutes the colour of the fur and of the eyes, Lyon 1955) hereditary absence of one or several

otoliths occurs frequently while the maculae as such and all the other parts of the labyrinth are apparently perfectly normal

Inner ear malformation comparable to those found in *Kreislers* and the type I of *Drehers* but com

meatus were found by A

atresia whose mothers had been injected subcutaneously before and during pregnancy with 1 cc of a watery solution of trypan blue

In another untreated rat (Altmann 1957) there was a severe malformation of the brain with absence of the corpus callosum and marked hydrocephalus The ears showed identical findings on both sides The external ear and the drum were normal the malleus well developed but the tensor tympani muscle absent The incus was rudimentary the stapes oval window styloid process and stapedial muscle were absent The utricle and saccule were again very wide and communicated with each other through a wide opening both maculae were present The endolymphatic duct and sac were absent the semicircular canals normal The cochlear duct was transformed into a slightly curved cystic sac which showed on its postero medial circumference a typical organ of Corti and a stria vascularis The cochlear aqueduct was absent

A review of the above described labyrinthine anomalies in rats shows that they can be divided according to the severity of the changes into the following four groups *Malformations of the entire labyrinth though of varying degree* with either dilatation of certain portions of the endolymphatic spaces (Shaker shorts Kreislers Drehers type I Rats described by Altmann) or with compression of the endolymphatic spaces (Dreher type II) malformations of the semicircular canals alone (Twirlers Zigzag) malformations of the maculae (absence of otoliths Pallids) progressive collapse of the originally well formed endolymphatic system (Shakers with Syndactylism)

In all instances with exception of the rats described by Altmann were the external and middle ears normal

In Man malformations of a severity comparable to that present in the shaker shorts Kreisler s Dreher s type I and the three rats described by the authors are rare

A still more severe malformation combined with left sided atresia of the external auditory meatus and bilateral microtia anomalies in the structure of the cerebral cortex spina bifida sacralis and bilateral clubfeet was recently reported by Schwarz (1962)

A *microcephalic fetus of six months* examined by Fleischer (1953) with defect of the eyes the nose the forebrain and cerebellum and with atresia ani showed a very severe malformation of the left inner ear

The right side was less severely malformed Utricle and saccule formed a wide common sac with a well differentiated macula Only the two vertical semicircular canals were present The cochlear duct was represented by a cystic sac which in some places showed a stria like structure The endolymphatic duct and sac were evidently absent

Of considerable clinical significance is a group of still less severe malformations affecting the cochlear duct, the osseous framework of the cochlea and the saccule. They are regarded as the anatomical basis of the so called hereditary degenerative type of impaired hearing and deafness which is assumed to show a dominant hereditary trait.

Siebenmann named these cases "Typus Mondini" because they show essentially the same structure as a case microscopically described by Mondini (1791) and a case microscopically described by Alexander (1904).

The bony capsule is flattened from the base to the apex and the normal arrangement of the cochlea can be seen only in the basal coil where the septum between lower basal and middle turn, originating from the cartilaginous capsule, is present. In the upper parts the interscalar septa, formed through intra-membranous ossification within the connective tissue, were absent and a scala communis or cloaca has been formed. The modiolus and the spiral laminae also show defects of varying degree. The cochlear duct often presents a reduction in the number of turns to two or even to one and one half. Its lumen is in some instances markedly dilated either along its entire course or in its upper parts only. In other cases the duct is collapsed. The saccule is often dilated, sometimes collapsed; the endolymphatic duct and sac are as a rule dilated (Secretan).

The utricle is only occasionally dilated; the semicircular canals are as a rule normal.

The organ of Corti is usually absent in part of the duct, particularly in the more malformed upper region, and reduced to a flat mound of more or less undifferentiated cells in the lower region. The stria vascularis and the spiral ligament are often atrophic. Frequently the spiral ganglion shows a reduction in the number of the ganglionic cells and of the nerve fibers belonging to the more affected upper parts. In rare instances the spiral ganglion is completely absent. In other cases it is situated in the central parts of the modiolus and in the fundus of the internal auditory meatus. The cochlear division of the eighth nerve is often abnormally thin.

Cases of the Mondini type of inner ear malformation occur not only in Man but also in dogs (Alexander & Tandler). A bilateral malformation of the just described kind but somewhat more marked was recently reported by Fleischer (1953) in a deaf mute woman of 31 years.

The cochlea showed a typical Mondini type of malformation; the saccule, utricle and endolymphatic were dilated. No mention is made of the endolymphatic sac. In addition the membranous as well as the osseous lateral semicircular canal was markedly dilated. The epithelium of the maculae was flattened; the cristae ampullares were well formed.

Inner ear malformations of varying severity are occasionally found in ears with congenital atresia of the external auditory meatus (see Altmann 1933, Case [illegible]).

Inner ear malformations, mostly of lesser degree, are common in anencephali.

In a newborn anencephalus examined by Fleischer (1953) there was a Mondini type of malformation of the cochlea without any changes in the vestibule and in the semicircular canals. No mention is made of the endolymphatic duct and sac. In other instances the duct and sac were found diffusely dilated (Agazzi, Nager, 1953). In three anencephali examined by Altmann (1957) there were only less marked malformations of the cochlea. In other cases reduction in length of the cochlear duct was accompanied by dilatation of the cochlear duct (Agazzi) or even by a diffuse dilatation of the ductus and saccus endolymphatics (Nager). In some instances rudimentary development or complete absence of one or two semicircular canals has been observed (see Altmann, 1957).

Lately, the roentgenological, particularly the tomographic examination of the inner ear has in a number of cases with as a rule unilateral deafness demonstrated labyrinthine changes *in vivo* which can best be interpreted as developmental anomalies (Psenner, Portmann and Guillen, Everberg, Rossberg *et al.*).

Recently we had the opportunity to study another human case with marked inner ear pathology.

CASE REPORT

Case 2

Colored female fetus, 35 weeks old, birth weight 2570 grams. Died one *hour and 20* minutes after birth. The mother is a 26 year old Gravida III, *Para II*, with blood group O, Rh negative, serology negative, whose other *two living children* were *full* term without exchange transfusions. The father's blood group is *O, Rh positive*. The pregnancy was complicated by polyhydramnios.

Autopsy diagnosis (Babies Hospital, A 9550) *was in part* as *follows*: Prematurity, spina bifida with ruptured meningocele mid thoracic area (below Th 5). Hydrocephaly, internal, *marked*. *Arnold Chiari* syndrome (elongated medulla oblongata, herniation *of small cerebellum* into foramen occipitale magnum). Erythroblastosis fetalis, moderate. (Hepatosplenomegaly.)

Histological examination of the temporal bones (serial sections 20 μ thick, stained with Hematoxylin *Eosin after* fixation in formalin, decalcification and embedding in celloidin.

Right side: horizontal sections

External and middle ear without pathology, the course of the facial nerve is normal.



Fig. 6. Case 2. Right side. View of utricle (*u*), saccule (*s*), cochlear duct (*d*) and ampullate end of lateral semicircular canal (*l*). Folds protruding into the saccule and the cochlear duct (*f*). Utriculo-saccular opening (*o*).

Inner ear

Pars superior. The vestibule shows normal size. The *utricle* is very wide, contains pink staining granular material and communicates through a fairly large opening with the *saccule*. A distinct utricular duct is missing (Fig. 6). The macula is of normal size and well supplied with nerve fibers but poorly preserved; some of the hair cells and supporting cells are still recognizable but not the otolithic membrane. The lumen of the *bony lateral semicircular canal* is considerably wider than under normal conditions. The *membranous lateral canal* is more than 10 times wider but much shorter than the normal canal. It fills about one half of the lumen of the bony canal. The ampulla is still wider and the *crista ampullaris* reduced to a flat hill. The epithelium shows autolytic changes but some hair and supporting cells can still be recognized. The cupula is detached and floating in the lumen of the utricle. The nerve supply is normal.

The lumina of the bony *vertical canals* are normal in size, the *membranous canals* about 6 times wider than the normal canals and also contain pink staining granular material. The lean ends of the two vertical membranous canals converge and eventually merge but the *common crus* instead of going from this point into the utricle ends blindly.

The *crista ampullaris* of the superior vertical canal (Fig. 7) is completely



Fig 7 Case 2 Right side Superior vertical canal with flattered crista ampullaris (*a*) Macula utriculi (*u*)



Fig 8 Case 2 Right side Crista ampullaris of posterior vertical canal (*a*) and accessory crista (*aa*)



Fig 9 Case 2 Right side Crista quarta (q) in the medial wall of the utricle and papillary excrescences (p) in the utricular and saccular wall near the utriculo saccular opening

flattened out but well supplied with nerve fibers Some hair and supporting cells are still recognizable but the cupula is absent

The shape of the *crista ampullaris* of the *posterior vertical canal* is more or less normal but the cupula again detached and floating in the lumen In addition to the regular crista which is well supplied with nerve fibers a smaller *accessory crista* is found adjacent to but somewhat farther away from the vestibule than the normal crista (Fig 8) It shows the same structure as the normal crista but is supplied by a few nerve fibers only

In the lower part of the medial wall of the utricle near its communication with the saccule a low ridge extends downward to the floor Its side walls are covered with cuboideal epithelium its crest with columnar epithelium in which due to the poor preservation no further details can be made out The crest of the ridge had apparently been covered with a cupula which is now detached from the underlying epithelium (Fig 9) The stroma consists of loose connective tissue with numerous thin walled small blood vessels The subepithelial layer contains many branched pigmented cells but no nerve fibers In a circumscribed area adjacent to the opening of the utricle into the saccule the antero medial wall of the utricle shows papillary excrescences which are covered with low columnar epithelium (Fig 9)

Pars inferior The saccule is very wide its macula poorly preserved but its structure and nerve supply normal The lumen again contains a small amount of fine granular material In some places the wall of the saccule is

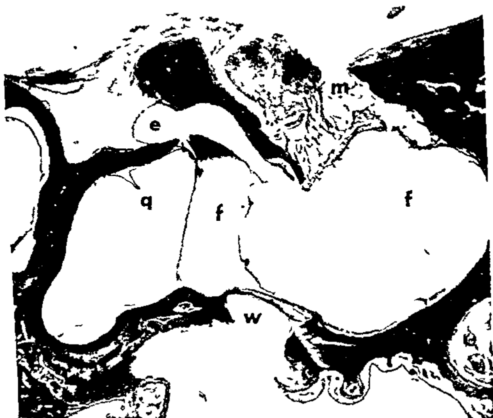


Fig 10 Case 2. Right side Wide and short endolymphatic duct and sac (*s*). Folds protruding into the saccule and the cochlear duct (*f*). Crista quarta (*q*) round window niche (*w*) internal auditory meatus (*m*)

folded inward (Fig 6) A particularly large vertical fold extends from the lateral saccular wall near its transition into the posterior wall inward, almost completely covers the opening between the utricle and the saccule (Fig 6) and protrudes into the saccular and the endolymphatic duct (Fig 10) The reunient duct is not recognizable and the saccule is in wide communication with the very wide cochlear duct (Fig 6)

The saccule continues without distinct border into a very wide *saccular duct* and the latter into the wide and short *endolymphatic duct* The *endolymphatic sac* is also wide and shorter than usual (Fig 10) It is lined with the same flat epithelium as the duct, with exception of a few niches where the epithelium is more cuboideal (Fig. 11)

The postero superior wall of the saccule shows in a small circumscribed area near the opening into the utricle and adjacent to the antero medial wall of the utricle the same papillary excrescences covered with low columnar epithelium which were already described in the utricle (Fig 9) The round window membrane is well developed, the scala tympani reduced to a narrow partly obliterated slit (Fig. 10) The aquaeductus cochleae is absent

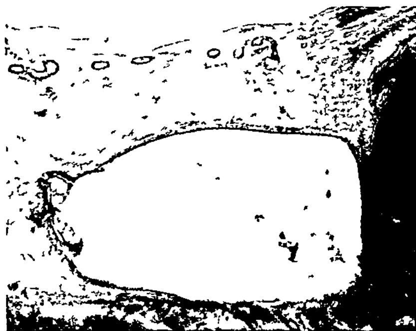


Fig 11 Case 2 Right side Endolymphatic sac

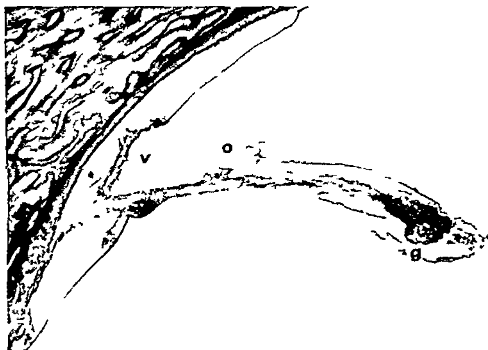


Fig 12 Case ... Upper half of basal cochlear turn Organ of Corti (o) stria vascularis (v) cochlear ... ganglion cells (g)



Fig 13 Case ? Right side Prolapse of the dilated cochlear duct (*d*) through the tractus foraminosus into the fundus of the internal auditory meatus

The *cochlear duct* describes about two turns The basal turn is considerably dilated but partly collapsed the upper turn somewhat less dilated A large fold protrudes from the anterolateral wall in a postero medial direction into the lumen (Fig 6 10) The tractus foraminosus contains some thin bony trabeculae in its peripheral portion the central part is composed of a thin layer of connective tissue modiolus interscalar septa and lamina spiralis ossea are completely absent The cochlear lumen is transformed into a so called scala communis or cloaca which is almost completely filled out by the dilated cochlear duct (Fig 19) The ligamentum spirale and the lamina spiralis membranacea protrude from the lateral cochlear wall into the cochlear lumen (Fig 12)

The *cochlear nerve* is thin and ascends covered with a sheath of connective tissue from the tractus foraminosus along the free inner margin of the spiral lamina from the basal to the upper turn contains some ganglionic cells and supplies the organ of Corti with nerve fibers The organ of Corti and the stria vascularis are markedly autolytic They are however well developed in the upper turn and the upper half of the basal turn less well recognizable in the lower half of the basal turn (Fig 12) The scala tympani has been almost completely obliterated and the scala vestibuli reduced to a narrow space above the upper cochlear turn The dilated cochlear duct has in a small area of the central portion of the tractus foraminosus broken through the connective tissue barrier and protrudes slightly into the fundus of the internal auditory meatus (Fig 13)



Fig. 13. Case 2. Left side. Bony obliteration of the lumen of the superior vertical canal (b).

Left side: vertical sections

External and middle ear show no pathology; the facial nerve takes its normal course.

Inner ear

Pars superior. The utricle is very wide, the macula of normal size and evidently normal structure but markedly autolytic; the nerve supply is normal.

The *lateral semicircular canal* including the crista is absent.

The *vertical canals* are only partially present. The crus ampullare and the ampulla of the *posterior vertical membranous canal* are again much wider than under normal conditions and fill the lumen of the bony canal almost completely. The crista is markedly autolytic and the cupula absent. The nerve supply is normal. The membranous canal takes its normal course and unites with the lean end of the superior vertical canal to form the common crus. However, only a very short portion of the latter is present and terminates blindly towards the vestibule. The *superior vertical membranous* canal is just as wide as the posterior canal. It can be followed upward and forward for a short distance from the site of union with the posterior canal and then ends blindly (Fig. 14). The ampulla of the superior canal has



FIG 15 Case 2 Left side Bony obliteration of the lumen of the ampullated end of the superior vertical canal (*b*)

evidently become incorporated into the dilated utricle and there is no distinct border between the two structures The crista is again markedly autolytic, the cupula absent but the nerve supply normal The part of the membranous canal between the ampulla and the blind end of the distal portion is absent The part of the ampullated end of the bony superior vertical canal adjacent to the ampulla is still recognizable but the lumen of the perilymphatic space has been almost completely filled with *bone* (Fig 15)

The *utricular* and *saccular ducts* are absent and the *utricle* communicates through a wide opening with the saccule

A *very narrow rudimentary endolymphatic duct and sac can be* seen within the bone of the petrous pyramid Its blind *proximal end* lies close to the vestibule (Fig 16) its wider distal end *close to the dura of the* posterior fossa Its wall forms low folds which are *covered with cuboidal* epithelium and protrude into the lumen (Fig 17)

Pars inferior The *saccule* is *also very* [illegible] again markedly autolytic The *nerve sup*[illegible] with its membrane and the *cochlear sup*[illegible] of the perilymphatic *space of the vestibule is very* greatly reduced

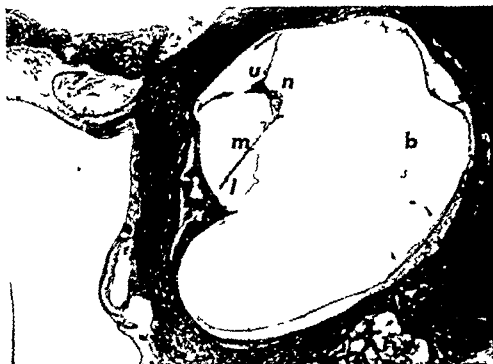


Fig. 19. Case 2. Left side. Cochlea with cochlear duct maximally dilated in the lower basal turn (*b*), somewhat less dilated in the upper turn (*u*). Cochlear nerve (*n*), spiral ligament (*l*), basilar membrane (*m*).

semicircular canals very wide. *lateral* canal short, common crus of the *vertical canals* almost completely missing. *Low ridge* in the lower part of the medial wall of the utricle with a cupula-like structure. *papillary excrescences* in the antero-medial wall of the utricle adjacent to the utriculo-saccular opening. *Accessory crista ampullaris* in posterior vertical canal.

Saccule very wide with folds protruding deeply into the lumen. *Papillary excrescences* in the postero-superior wall near the utriculo-saccular opening. *Endolymphatic duct* wide and short, originating from the saccule; *endolymphatic sac* wide and short, mostly lined with flat epithelium.

Cochlear malformation of the Mondini type with the cochlear duct wider in the basal than in the upper turn. Small herniation of the cochlear duct into the fundus of the internal auditory meatus. *Absence of the cochlear aqueduct. Cochlear nerve thin, vestibular* nerve well developed.

Left side. *Utricle* and *Saccule* very wide, communicating through a wide opening, utricular and saccular ducts absent. *Lateral membranous semicircular canal* including the crista absent. *Vertical canals very wide*, midportion of the *superior* vertical canal absent, its ampulla and crista incorporated into the utricle. *common crus* of the vertical canals almost completely absent. *endolymphatic sac and duct* hypoplastic, the proximal portion of the latter absent.

Cochlear malformation of the Mondini type with inward displacement of the tractus foraminous and herniation of the cochlear duct into the fundus of the internal auditory meatus *Cochlear nerve* thin, *vestibular* nerve well developed

COMMENT

The just described inner ear malformations show close resemblance to the findings in Kreisler—and Dreher type I—mice Even the herniations of the dilated basal turn of the cochlear duct into the fundus which so far had never been described in human specimens were present Aside from that the findings are also very similar to those reported previously by the author in two cases of congenital atresia of the external auditory meatus (Altmann 1933 case 2 lt side and case 8) and to the already mentioned findings of Fleischer (1953) in a deaf mute woman

The malformations are obviously the result of disturbances in the sub division of the otocyst into the three main portions, recessus labyrinthi (endolymphatic duct and sac) pars superior (utricle and semicircular canals) and pars inferior (saccule and cochlear duct) which starts during the fifth week of embryonic life From experiments in amphibians it has been known that intactness of the recessus labyrinthi is an essential factor for the proper differentiation of the otocyst otocysts which fail to develop a ductus or saccus endolymphaticus may swell and remain vesicular Intactness of the endolymphatic appendage is however no guarantee for normal differentiation of the otocyst The endolymphatic duct and sac were hypoplastic dilated and evidently non functional on the right side and severely hypoplastic and without connection with the main portion of the membranous labyrinth on the left side

The mechanism by which the dilatation of the endolymphatic spaces is brought about is still unknown It is evidently the result of disturbances in the fluid balance of the inner ear starting at an early stage of embryonic development long before the areas of future production and resorption of the fluids have become differentiated It is therefore not very probable that the fluids contained in the endo and perilymphatic spaces at that time have already the same chemical composition as the later endo and perilymph For that reason alone it seems hardly permissible to try to explain the labyrinthine changes on the basis of our concepts of the physiology of the labyrinthine fluids in fully developed individuals or to draw conclusions from the findings in malformed embryos regarding the normal physiology of the fluids

The changes in the semicircular canals are due to disturbances in the pouches from which in the 6th week of embryonic life the lateral and the two vertical semicircular canals develop Most probably the pouches were

already wide before their walls became adherent to each other at certain places and were subsequently resorbed while the persistent portions formed the abnormally wide semicircular canals.

On the right side the shortness of the lateral canal can be explained by hypoplasia of the pouch from which it is derived; the absence of the common crus of the vertical canals is due to excessive adherence and resorption of the walls of the common pouch for the two vertical canals in the area where the common crus should be formed.

On the left side the pouch for the lateral canal had failed to develop while the absence of the midportion of the superior vertical canal and of almost the entire common crus can again be explained by excessive adherence and subsequent resorption of the walls of the common pouch for the two vertical canals. The low ridge in the right utricle bearing a cupula-like structure represents a so-called crista quarta (Benjamins) or papilla neglecta (de Burlet), a rudimentary formation found in all vertebrates from fish to man but more frequently in other mammals than Man. It is regarded as the homologon of the macula neglecta described by Retzius in fishes, reptiles and birds (for details see Altmann 1953, Greck 1961).

The papillary excrescences in the right utricular and saccular walls near the utriculo-saccular opening are evidently identical with the papillary epithelial proliferations observed occasionally in young human embryos at the place where the endolymphatic duct originates from the utricular and saccular ducts. Apparently they represent remnants of small diverticula which are seen during early embryonic stages (Kolmer).

An accessory crista ampullaris is a most unusual finding in Man and no reference about it was found in the literature. However Werner (1960) mentions that in the cyclostome petromyzon the cristae of the two vertical semicircular canals are divided along a longitudinal axis into two portions and that a subdivision of the cristae has also been observed in certain birds.

Absence of the cochlear aqueduct, a very rare malformation, was seen by Altmann (1933) in a case of bilateral Mondini type of inner ear malformation associated with atresia auris congenita (case VIII, rt. side) and by Nager (1925).

The bilateral cochlear malformation of the Mondini type in this case is insofar atypical as the lower and not, as usual, the upper cochlear turn shows the more severe changes. Dilatation of the saccule, of the endolymphatic duct and sac is almost the rule in this type of malformation and [illegible] assumed a primary malformation of the sac as cause of the entire [illegible] with a disturbance in the resorption of the endolymph with [illegible] of the sac and of the parts above it. However it has [illegible] (Altmann 1950) that this assumption can hardly be [illegible] because [illegible] rease in the endolymphatic pressure would have [illegible] formation of the osseous labyrinthine capsule this would ha[illegible] been [illegible] the time of differentiation of the endolymph producing s[illegible]tures.

It is therefore more likely that the dilatation of the various parts is due primarily to an abnormal differentiation of the otocysts with excessive growth of the affected parts

A disturbance in the resorption of the endolymph in the endolymphatic sac even if present would not satisfactorily and invariably explain the dilatation of the higher parts Even complete destruction of the sac in monkeys (Lindsay) and in cats (Lindsay Schuknecht Neff and Kimura) has failed to produce changes in the saccule and the cochlear duct This failure is explained by the results of the morphologic studies of Saxen in dogs and Man and of experiments of Altmann and Waltner in rabbits cats, and monkeys which favour the assumption that endolymph is produced as well as resorbed in the same segment of the cochlear duct without under physiologic conditions flowing down into the endolymphatic sac as it had been assumed by Guild in guinea pigs Similar ideas were expressed by Naftalin and Harrison by Dohlman and Ormerod and recently by Lawrence and co workers who speak of a radial flow of the endolymph in contra distinction to the longitudinal flow postulated by Guild

The folds protruding into the right saccule and cochlear duct are more likely to represent fixation artefacts than intra vitam changes comparable to those described in the type two of the *Dreher* mutation of mice, although this possibility cannot be ruled out completely

In an attempt at getting a better understanding of the formal genesis of a given malformation it has been customary to determine its teratogenetic termination period i e the latest developmental stage at which the damaging factor must have been operative

In the just described malformation thus would at first sight have been, as already mentioned the 5th week of embryonic life (8 9 mm embryos) the time when the subdivision of the otocyst in its three compartments had started However a closer analysis of the factors determining the course of the normal embryonic development shows that the developmental disturbance must have started at a much earlier time In this connection the association of the malformation of the inner ears with malformations of the brain and the spinal cord with the so called Arnold Chiari malformation (see Barry

ality of the blastopore upon the notochord the paraxial mesoderm (vertebral column and ribs) and the intermediate mesoderm (nephrogenic cord) combination of the Arnold Chiari syndrome with inner ear malformations has never before been described

The results of experimental work on the mechanics of development of the head region in amphibia which were reviewed previously (Altmann 1957) show that two main growth areas can be distinguished there (1) The prosencephalic or rostral area which comprises forebrain midbrain nose and eyes and which is underlaid by the anterior part of the parachordal

already wide before their walls became adherent to each other at certain places and were subsequently resorbed while the persistent portions formed the abnormally wide semicircular canals.

On the right side the shortness of the lateral canal can be explained by hypoplasia of the pouch from which it is derived; the absence of the common crus of the vertical canals is due to excessive adherence and resorption of the walls of the common pouch for the two vertical canals in the area where the common crus should be formed.

On the left side the pouch for the lateral canal had failed to develop while the absence of the midportion of the superior vertical canal and of almost the entire common crus can again be explained by excessive adherence and subsequent resorption of the walls of the common pouch for the two vertical canals. The low ridge in the right utricle bearing a cupula like structure represents a so called crista quarta (Benjamins) or papilla neglecta (de Burlet) a rudimentary formation found in all vertebrates from fish to man but more frequently in other mammals than Man. It is regarded as the homologon of the macula neglecta described by Retzius in fishes, reptiles and birds (for details see Altmann 1933, Gacek, 1961).

The papillary excrescences in the right utricular and saccular walls near the utriculo-saccular opening are evidently identical with the papillary epithelial proliferations observed occasionally in young human embryos at the place where the endolymphatic duct originates from the utricular and saccular ducts. Apparently they represent remnants of small diverticula which are seen during early embryonic stages (Kolmer).

An accessory crista ampullaris is a most unusual finding in Man and no reference about it was found in the literature. However Werner (1960) mentions that in the cyclostome petromyzon the cristae of the two vertical semicircular canals are divided along a longitudinal axis into two portions and that a subdivision of the cristae has also been observed in certain birds.

Absence of the cochlear aqueduct, a very rare malformation, was seen by Altmann (1933) in a case of bilateral Mondini type of inner ear malformation associated with atresia auris congenita (case VIII, rt side) and by Nager (1925).

The bilateral cochlear malformation of the Mondini type in this case is insofar atypical as the lower and not as usual the upper cochlear turn shows the more severe changes. Dilatation of the saccule, of the endolymphatic duct and sac is almost the rule in this type of malformation and S... re... assumed a primary malformation of the sac as cause of the entire ... sturbance in the resorption of the endolymph with ... the sac and of the parts above it. However it has ... it (Altmann, 1950) that this assumption can hardly ... r... rease in the endolymphatic pressure would have ... n... f the osseous labyrinthine capsule, thus ... tim... of differentiation of the endolymph

★

PRINTED IN SWEDEN BY

Almqvist & Wiksells Boktryckeri Aktiebolag

UPPSALA 1964

plate (2) the deuterencephalic or caudal area which concerns us here and which gives rise to rhombencephalon, labyrinth and branchial gut it is underlaid by the posterior part of the prechordal plate and the chorda mesoderm of the archenteric roof

Disturbance in the deuterencephalic growth area which achieves its final determination during neurulation are therefore more likely to be responsible for the malformation of the hindbrain as well as of the ears This would push back the teratogenetic termination period from the 6th embryonic week to a much earlier date

Several factors seem essential for the development of normal ears The ear anlage must develop at the proper place if this is the case the chorda mesoderm comes to lie under the prospective ear ectoderm during gastrulation and the raising of the neural folds brings the hindbrain in relation to the ear ectoderm

The mesoderm seems to be the primary inductive factor The mesodermal activation grows in intensity until the neural folds start to form, then diminishes and eventually completely disappears

It is followed by neural activation which is most effective during the process of fusion of the neural folds and then decreases in intensity The further normal differentiation of the otic vesicle is thought by many authors to be contingent on the presence and intactness of the medulla oblongata There is however evidence that the medulla is not necessary for late differentiation of the otocyst

Another essential factor as already mentioned seems to be the intactness of the endolymphatic appendage

There are strong indications that the development and differentiation of the otic vesicles in mammals is governed essentially by the same factors as in amphibians Consequently the findings in Kreislers (Hertwig 1944) and possibly also in Drehers (Fischer 1956) were explained as the result of an invagination of the otic pit at a greater distance from the neural tube than in normal embryos with a subsequent diminution of the inductive influences acting upon the ear anlage

The close similarity between the ear malformations in Drehers and those in a group of human malformations representing the so called hereditary degenerative type of deafness with dominant hereditary trait (see Albrecht 1940) had already been stressed by Fischer

It is quite probable that the human ear malformation described in this paper is due to the same cause to formation of the otic placodes at a greater distance from the neural tube than in normal embryos If this assumption is correct the teratogenetic termination period would be the 2 3 somite stage the stage at which the otic placodes sometimes already become recognizable However the question why they are formed at the wrong place still remains open It could be the result of abnormal inductive influences emanating from the chorda mesoderm an assumption which would also explain the combination of the ear—with brain malformations Under these circum

REFERENCES

AGAZZI, C Les altérations de l'oreille dans l'anencephalie *Pract oto-rhino-laryng 8* 114, 1946

ALEXANDER, G Zur Pathologie und pathologischen Anatomie der kongenitalen Taubheit *Arch f Ohrenheilk 61* 183, 1904

— Pathologische Anatomie der nervosen Anteile des Gehororganes in *Handbuch der Neurologie des Ohres*, ed by G Alexander and O Marburg, vol 1 Berlin Urban & Schwarzenberg, 1924, p 701

ALEXANDER, G, and TANDLER, J Untersuchungen an kongenital tauben Hunden, Katzen und an Jungen kongenital tauber Katzen *Arch f Ohrenheilk 66* 161, 1905

ALTMANN, F Zur Anatomie und formalen Genese der Atresia auris congenita *Monatschr f Ohrenheilk 76* 765 1933

ALTMANN, F Histologic picture of inherited nerve deafness in Man and animals *Arch Otolaryngol 51* 852, 1950

ALTMANN, F Malformations of the auricle and the external auditory meatus *Arch Otolaryngol 54* 115, 1951

— Malformations, anomalies and vestigial structures of the inner ear *Arch Otolaryngol 57* 591, 1953

— Congenital atresia of the ear in Man and animals *Annals Otol, Rhin & Laryngol 64* 824 1955

— The ear in severe malformations of the head *Arch Otolaryngol 66* 7, 1957

ALTMANN, F & WALTNER, J G Further investigations on the physiology of the labyrinthine fluids *Annals Otol, Rhinol & Laryngol 59* 657 1950

BARRY A PATTEN, B & STEWART, B Possible factors in the development of the Arnold Chiari malformation *Journ Neurosurg 14* 285 1957

BENJAMINS E C. Beitrag zur Kenntnis des hautigen Labyrinthes *Zeitschr f Ohrenheilk 68* 101, 1913

BIERWOLF D Kleinhirnmissbildungen durch hereditaren Hydrocephalus bei der Hausmaus *Wissenschaftliche Zeitschr d Martin Luther Universitat Halle Wittenberg 5* 1237 1956

BONNEVIE K Abortive differentiation of ear vesicle following hereditary brain anomaly in short tailed waltzing mice *Genetics 18* 105 1936

BUCH N H and JORGENSEN M B Pathological studies of deaf mutes *Arch Otolaryngol* 246 1963

DE BURLET H M Über die Papilla neglecta *[illegible] 66* 1[illegible] 1928

Vergleichende Anatomie des stato akustischen Organes in *Handbuch der vergleichenden Anatomie der Wirbeltiere* ed by Bolk Goppert Kallius & Lubosch v 1 2 pt 2 Berlin Urban & Schwarzenberg 1934 p 1293

CAMERON A H Malformations of the neurospinal axis urogenital tract and foregut in spina bifida attributable to disturbance of the blastopore *Journ Path Bact* [illegible] 413 [illegible]

CANTELI P G Studio anatomo microscopico di malformazioni dell orecchio in un caso di idrocephalo congenito con cheilo-gnato urano schisi laterale *Atti Soc med-chir Padova 11* 913 1934

DEOL M S The anatomy and development of the mutants pirouette shaker 1 and waltzer in the mouse *Proc Roy Soc B 145* 206 1956

DOHLMAN G and ORMEROD F C The secretion and absorption of endolymph *Acta otolaryngol 51* 435 1960

EVERBERG G Investigations into unilateral total deafness and absence of vestibular function with a particular view to the X ray appearances in the inner ear *Acta otolaryngol 52* [illegible]

EVERBERG G *Ensidig dovhed* Kobenhavn Christtreus Bogtrykkeri [illegible]